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DIABETIC COMA,—THEN AND NOW*

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INTRODUCTION

IN 1916, in the first edition of his "Treatment of Diabetes Mellitus" Joslin[1] wrote: "For many years my attitude toward coma was to consider it the culmination of the diabetes, and after the death of the comatose patient I could honestly unite with the family, the physician and the nurse in feeling that no more could have been done. But for me this comfortable creed has passed, and in its place has come the opinion that coma by no means represents the culmination of the disease, that it is not a justifiable accident, and, though not in all, yet in most cases, it is avoidable."

DIABETIC COMA BEFORE INSULIN

Up to October 1922, when insulin was first used clinically, there had entered the medical wards of the Peter Bent Brigham Hospital some seventeen diabetic patients with sufficient acidosis to produce a diminution of the blood bicarbonate concentration to a point below 25 volumes per cent. and a clinical picture of marked air hunger with more or less complete unconsciousness. These patients were treated in various ways, chiefly with large amounts of fluid. Many received infusions of glucose, levulose, or bicarbonate of soda. All of them died within five days of their entry to the hospital. No complication of any other common chronic illness had so complete a mortality.

Case No.	Age	Sex	Duration of diabetic symptoms before coma	Preliminary symptoms of coma	Days in Hospital	Outcome of coma
1.	7	F	2 months	Sudden onset of coma without warning.	1 day	Death
2.	13	M	6 months	Sudden onset of coma without warning.	1 day	Death
3.	14	M	1 month	Vomiting followed by coma.	1 day	Death
4.	18	M	1 year	Vomiting followed by coma.	3 days	Death
5.	25	F	Not known	Sudden onset of coma without warning.	1 day	Death
6.	26	F	1 year	Vomiting followed by coma.	1 day	Death
7.	27	F	2 years	Acute respiratory infection followed by coma.	3 days	Death
8.	28	M	2 years	Acute respiratory infection followed by coma.	2 days	Death
9.	30	M	1 year	Sudden onset of coma without warning.	3 days	Death
10.	36	M	1 year	Vomiting followed by coma.	3 days	Death
11.	39	M	1 month	Vomiting followed by coma.	1 day	Death
12.	41	F	1 year	Vomiting followed by coma.	4 days	Death
13.	43	F	2 years	Vomiting followed by coma.	5 days	Death
14.	46	M	Not known	Acute respiratory infection followed by coma.	1 day	Death
15.	53	F	2 years	Infected finger followed by coma.	2 days	Death
16.	58	M	2 months	Increasing weakness followed by coma.	2 days	Death
17.	59	F	12 years	Loss of appetite, vomiting followed by coma.	2 days	Death

FIGURE 1. Diabetic Coma Without Insulin.

At the Peter Bent Brigham Hospital there has been an opportunity to study a group of patients in diabetic coma treated by various methods. It is my purpose in this paper to review our experience with this symptom complex in order to contrast the diabetic coma of yesterday with that of today and to emphasize the soundness of Joslin's prediction. Nowadays no honest doctor can camouflage a fatal case of diabetic coma as representing the natural culmination of the disease, nor believe with a clear conscience that fatal diabetic coma is ever a justifiable or inevitable catastrophe.

Figure 1 summarizes the course of diabetic coma as many doctors saw it a decade ago. It was almost uniformly fatal. It occurred commonly in young people, the average age of our group being thirty-two years. It was seen, therefore, especially in those cases which were generally believed liable to run an acute downward course. But few of our patients were known to have had diabetic symptoms longer than a year, so that the average total duration of the disease was only about twelve months. Coma might appear to follow some trivial infection or it might, apparently, develop suddenly and out of a clear sky. The most constant symptoms, noted at the onset of a striking change in the character of the patient's breathing or mental reaction, were nausea and vomiting. An upset stomach was apt to be followed quickly by coma and death.

*From the Medical Clinic of the Peter Bent Brigham Hospital, Boston. Read before the Brockton Medical Society on October 10, 1929.

†For record and address of author see "This Week's Issue," page 1008.

THE FASTING TREATMENT OF ACIDOSIS

A certain number of diabetics with a considerable degree of acidosis came for treatment but thanks to Dr. F. M. Allen's[2] starvation program were able to survive. In my own experience, I recall in the pre-insulin days seeing but one patient who was able to overcome acidosis whose blood bicarbonate concentration reached a point lower than 15 volumes per cent., though with readings above 25 and 30 volumes per cent., recovery was not uncommon.

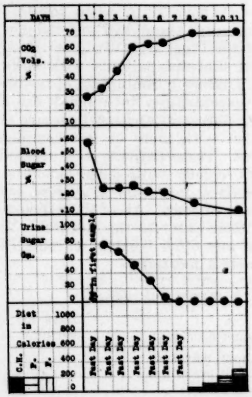


FIGURE 2. The Treatment of Diabetic Acidosis and Glycosuria Without Insulin.
A man, 22 years old. Diabetic symptoms for three weeks. Not in coma. Death four months after treatment was begun.

A patient with acidosis who was fasted successfully, became sugar-free in due course of time. The alkaline reserve of the blood plasma rose gradually to normal and the blood-sugar concentration fell. The immediate therapeutic result was striking, certainly, but it was no joke to induce an emaciated young person, hungry as a bear, to starve himself for a week or ten days. Such therapy required a good deal of fortitude and perseverance on the patient's part and a certain amount of boldness on the doctor's. Furthermore, the end-result of a long-continued course of malnutrition was often not too satisfactory. Of thirty-seven patients entering the Rockefeller[3] Hospital with severe acidosis, twenty-six (70%) were dead in less than two years. This experience was by no means uncommon. Most doctors in general practice were justified in feeling that an attack of severe diabetic acidosis was of very evil omen, since even if immediately survived, it was usually followed by death in a few months' time. Only a man of Dr. Joslin's optimism and sagacity could predict that the day would come when diabetic coma would no longer be considered the inevitable and perhaps fortunate culmination of that baffling illness called diabetes mellitus. Most of us felt that the treatment of severe diabetes was almost hopeless.

DIABETIC COMA AFTER INSULIN

Since October 1922, when insulin was first used clinically, there have entered the medical wards of the Peter Bent Brigham Hospital some twenty-three patients with sufficient acidosis to produce a diminution of the blood bicarbonate concentration to a point below 25 volumes per cent. and a clinical picture of marked air hunger with more or less complete unconsciousness. These patients have been treated in various ways, chiefly with insulin and large amounts of fluid. A few have received infusions of glucose or bicarbonate of soda. Of these cases, fifteen have recovered from the coma and eight have died within a few days of their entry to the Hospital. No complication of any other common chronic illness has ever responded to proper treatment in so spectacular a manner as does diabetic coma at the present time. Uncomplicated diabetic coma is no longer an especially serious illness.

Figure 3 summarizes the course of diabetic coma as many doctors see it today. It should not prove fatal and is amenable to treatment. It is still likely to develop in young diabetics who have had the disease for but a comparatively short time. Since, however, diabetes on the whole is recognized earlier and treated better than it was a few years ago, we now encounter more frequently older diabetics with coma or young people with diabetes of longer standing who, for one reason or another, have abandoned treatment and thus lapsed into coma. The complication may still appear to develop suddenly and out of a clear sky or may follow a trivial infection. The most constant symptoms at the onset of a striking change in the character of the patient's breathing or mental reactions continue to be nausea and vomiting so that an upset stomach is often followed by severe acidosis.

Figure 4 summarizes the course of our fatal cases. These cases are of especial importance because they illustrate two very important facts —namely, that a diabetic patient in coma who has also a severe infection is less likely to get well than a comatose case without infection. An adequate amount of insulin must be given in order to save life.

THE METHOD OF ACTION OF INSULIN IN COMA

In the treatment of acidosis, insulin accomplishes in a few hours what fasting accomplished in a few days. It is necessary, however, to use large and repeated doses of the drug and a modicum of intelligence. Under proper management the blood sugar level falls quickly to normal, the acidosis, as measured by the blood bicarbonate concentration, disappears very promptly, the sugar in the urine vanishes, the patient regains consciousness, begins to eat and drink normally and the emergency quickly passes over.

No longer is prolonged starvation or a protracted course of malnutrition necessary. An at-

Case No.	Age	Sex	Duration of diabetic symptoms before coma	Preliminary symptoms of coma	Outcome of coma
1.	13	F	4 years	Sudden headache followed by nausea, vomiting and coma.	Recovery.
2.	13	F	2 months	Sudden loss of appetite, nausea, vomiting and coma.	Recovery. Had one previous attack of coma and one or two subsequent attacks.
3.	13	M	1 year	Abdominal cramps followed by nausea, vomiting and coma.	Recovery. Has had three subsequent attacks of coma, one of which was complicated by gangrenous appendicitis.
4.	13	M	1 year	Abdominal pain with nausea, vomiting and coma.	Recovery.
5.	17	F	2 years	Sudden nausea and vomiting followed by coma.	Recovery. Had one previous attack of coma.
6.	20	M	6½ years	Abdominal pain with nausea, vomiting and coma.	Recovery.
7.	20	M	2 weeks	Sudden nausea and vomiting followed by coma.	Recovery.
8.	23	M	1½ years	Abandoned diet. Became short of breath and finally comatose.	Recovery. Had three subsequent attacks of coma.
9.	27	F	1 year	Abdominal pain with nausea, vomiting and coma.	Recovery.
10.	32	M	5 years	Abandoned insulin voluntarily. Nausea, vomiting and coma.	Recovery.
11.	34	M	4 years	Abandoned treatment. Nausea, vomiting and coma.	Recovery.
12.	34	F	8 months	Nausea, vomiting and coma.	Recovery. One subsequent attack of coma.
13.	40	F	2 years	Abandoned treatment. Became comatose and dyspnoeic.	Recovery.
14.	58	F	3 years	Abdominal pain. Nausea and vomiting.	Recovery.
15.	45	F	3 years	Sudden onset of coma.	Recovery. Complicated by empyema.

FIGURE 3. Diabetic Coma With Insulin. Cases Treated Successfully.

Case No.	Age	Sex	Duration of diabetic symptoms before coma	Preliminary symptoms of coma	Days in Hospital	Outcome of coma
1.	18	F	1½ years	Sore throat followed by coma.	1 day	Death. Bronchopneumonia at necropsy.
2.	21	F	7 months	Abandoned treatment. Vomiting followed by coma.	3 days	Death. No necropsy.
3.	22	F	1½ years	Sore throat followed by coma.	1 day	Death. Streptococcus in blood culture.
4.	28	M	3 years	Sudden onset of coma.	2 days	Death. Bronchopneumonia at necropsy.
5.	44	F	1 year	Sudden onset of coma.	2 days	Death. No necropsy.
6.	61	F	2 years	Sudden onset of coma.	1 day	Death. Died suddenly. No necropsy.
7.	64	F	9 years	Sore throat followed by coma.	1 day	Death. Bronchopneumonia at necropsy.
8.	65	F	8 years	Vomiting followed by coma.	4 days	Death. Our second case. Supply of insulin ran out. Patient lapsed into coma after beginning improvement.

FIGURE 4. Diabetic Coma With Insulin. Fatal Cases.

tack of coma is an acute affair, over with quickly, and is apt to be far less debilitating to the patient than is a simple appendectomy, or a quinsy sore throat to the ordinary individual.

THE PROGNOSIS OF DIABETIC COMA

A few years ago many doctors looked askance at modern tendencies in diabetic therapy. They

saw nearly all patients with diabetic coma die; the few that recovered from severe acidosis were kept alive by virtue of very rigid under-nutrition only to die a few months later. The treatment seemed almost too drastic to justify even the best results. Today, however, insulin convinces the most skeptical. Of our seventeen recovering cases, several, to be sure, have had repeated attacks of coma since we first saw them, yet all but five are now alive and well. One patient has died of tuberculosis and two from deliberate carelessness. We do not know the cause of death in the other two cases.

To compensate for these fatalities, one of our young girls has grown up and had a baby. Our living children are all doing splendidly at present, seeming to gain tolerance and to develop normally, and our adults get older year by year very gradually and with peculiarly little to complain of in relation to diabetes.

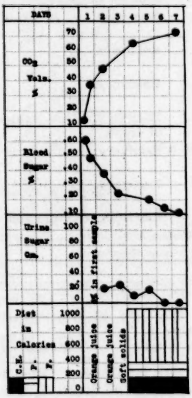


FIGURE 5. The Treatment of Diabetic Acidosis and Glycosuria with Insulin.

A woman, 24 years old. Diabetic symptoms for six months. In coma for 24 hours before treatment was begun. Alive and in good condition four years later.

Wilder and Adams[4] have followed up the first thousand diabetic patients coming to the Mayo Clinic after insulin was available. In this group, at entry to the Clinic, were ten patients with the complication of active pulmonary tuberculosis, twenty-five with gangrene and twenty-nine with sufficient acidosis to reduce the blood bicarbonate concentration to a point below twenty-five volumes per cent. Now seven of the ten patients with tuberculosis are dead (70%), sixteen of the twenty-five with gangrene (64%), and only ten of the twenty-nine with acidosis (34%). All of these patients have been followed for at least four years after they were first seen in Rochester and many for five or six years.

Such a follow-up record is by no means remarkable in a group of perfectly treated cases. In other words, in view of our present knowledge regarding the ideal treatment of diabetes and its final prognosis, the complication of coma appears to be of far less ultimate seriousness than either the complication of gangrene or tuberculosis. Today the patient with coma, barring infection or accident, gets well of his attack and thrives like a healthy person; yesterday's case of coma died with almost unfailing promptness or eked out a miserable existence for only a few months after the attack.

THE DIAGNOSIS OF DIABETIC COMA

The successful treatment of diabetic coma depends on its early recognition and this in large measure depends on the keenness of the family doctor. Any patient with diabetes may develop coma. Coma may come on suddenly or follow even the most minor infection. All patients with diabetes, therefore, may develop coma and must be carefully watched.

The patient bordering on diabetic coma is likely to have an upset stomach, and many complain of abdominal pain. The differential diagnosis between diabetic acidosis and an acute abdominal emergency in a patient with diabetes is by no means simple, especially since in both conditions there may be shock, fever or subnormal temperature, and an elevated white blood count. On general principles it is safer to assume that a diabetic patient, with sugar in the urine, pain in the abdomen, nausea and vomiting, a flushed face and air hunger has acidosis rather than appendicitis, and to use insulin and then to call the surgeon.

All older diabetics have vascular disease and not uncommonly involvement of the coronary vessels. It is striking in going over our records, to discover that both before and since the days of insulin there have been several cases of proved cardiac infarction complicated by glycosuria and acidosis. In each of these cases the problem had arisen as to whether or not the clinical picture observed was primarily due to the diabetes or the circulatory disturbance. When dealing with older diabetics, it is worth bearing in mind that the apparent coma may be referable in some manner to a cardiac complication, even though treatment for coma is indicated. A sudden exitus, however, usually can be traced to another illness than diabetes.

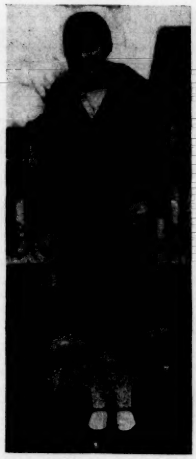
In the group of older patients, too, a cerebral vascular complication is sometimes encountered. A sudden, cerebral, vascular accident will throw a patient into coma. Glycosuria may be present, but in the treatment of this type of case insulin will not prove especially helpful. The important thing from the point of view of treatment and prognosis is to establish the correct diagnosis.

A doctor who undertakes the treatment of a patient with diabetes assumes a large responsibility. Not only must he recognize the disease but he must also follow up every detail of its treatment so long as his patient lives. A man or woman must possess self-control and philoso-

phy to bear diabetes with equanimity. A certain number abandon treatment deliberately and go downhill while others go from doctor to doctor hoping for a new method of cure. Much can be done by the proper person towards stabilizing morale and in leading patients to use insulin and diet to best advantage. This is one of the most important parts of today's treatment of diabetes.

I like Dr. Joslin's[5] rules for the prevention of coma. They are simple, direct and dogmatic. If all doctors were as familiar with them as are the Deaconess Hospital patients, there would be much less fatal diabetic coma in the future than there is at present. To prevent coma, Dr. Joslin advises his patients as follows:

1. Go to bed whenever indisposed.
2. Call the doctor.
3. Drink a glass of liquid each hour such as coffee, tea, broths, diluted orange juice or water oatmeal gruel.
4. Secure a nurse or at least someone to wait upon you and save your strength.
5. Move the bowels by enema, and, if liquids have not been retained by mouth, follow the enema by an injection of one pint of lukewarm water containing a teaspoonful of salt.
6. Keep warm.
7. Never omit insulin when the urine contains sugar.



Case 1.

Case 2.

Case 3.

FIGURE 6. The Prognosis of Diabetic Coma.

Case 1. Sixteen years old. Acute onset of diabetic symptoms with coma four years before this picture was made. Patient has had repeated attacks of coma.

Case 2. Eighteen years old. Onset of diabetic symptoms six years before this picture was made. First attack of coma five and a half years before the picture was made. Patient has had repeated attacks of coma.

Case 3. Sixty-three years old. Onset of diabetic symptoms eight years before this picture was made. Attack of coma five years before this picture was made. No subsequent attacks of coma.

THE TREATMENT OF DIABETIC COMA

The principles of treatment for diabetic coma are comparatively simple. The patient in coma needs insulin because he is excreting a large amount of sugar in the urine and has a large accumulation of blood-sugar and acidosis; he needs fluids because he is dehydrated; he needs warmth and the best of nursing care because he is somewhat shocked and in a state of more or less unconsciousness; he needs a doctor because he is face to face with a dire emergency and may lose his life if the doctor is not properly on the job.

The manner in which treatment is carried out varies in the different hospitals. At the Peter Bent Brigham Hospital our nurses and house-officers have received the following instructions for the management of diabetic coma:

1. As soon as the patient arrives place him in a warm bed.
2. Give an enema and obtain a blood and urine sample.
3. Give 1000 cc. of saline as a subpectoral at once.
4. Give 25 units of insulin at once.
5. Give insulin about every two hours thereafter until the urine becomes sugar-free, judging the dose by the amount of sugar present in

catheter urine specimens. When the urine becomes sugar-free continue to examine it at three-to-four-hour intervals using enough insulin to prevent the return of glycosuria and acidosis.

6. Let the patient have about 4000 cc. of fluid each twenty-four hours during the first few days in the form of sub-pectoral injections, rectal taps or fluids by mouth. The rectal tap should consist of 5% glucose in saline or tap water. Do not use sodium bicarbonate.
7. As soon as the patient becomes co-öperative, let him have small amounts of warm fluids to drink, and, by way of food, as much orange juice, ginger ale, or oatmeal gruel as he likes.
8. As soon as the patient's condition warrants it, allow him to eat a low calorie "soft" diet including milk, cream, butter, crackers, eggs and cereals. Thereafter when the patient's stomach is settled, treat the diabetes as usual, remembering that cases near coma require large doses of insulin, and that as they improve, they regain tolerance rapidly and become increasingly liable to hypoglycemic reactions.
9. Comatose patients whose acidosis does not improve, as measured by the plasma bicarbonate, within eight hours after insulin is first given, should receive 25 gm. of sodium bicarbonate by mouth, rectum or vein during the course of a few hours. This dose should be repeated in twelve hours if acidosis persists.
10. Diabetic patients admitted to the Medical Wards with any complication or a possible surgical nature (carbuncle, gangrene, lymphangitis, etc.) should be seen by the Surgical Resident at once. A diabetic patient can be prepared for operation in a few hours by the proper use of insulin.
11. Hypoglycemic reactions are to be treated with the oral or rectal administration of ten to twenty gm. of sugar. Intravenous injections of sugar are rarely needed.

At the Deaconess[6] Hospital where their experience in the management of coma is greater than ours at the Peter Bent Brigham Hospital, larger doses of insulin are used and these are given at more frequent intervals. Forty or fifty units may be given at a dose and repeated every thirty minutes according to the condition of the urine and blood. Caffein sodium benzoate, in seven and one-half grains' dosage, administered subcutaneously, is used to stimulate the circulation. The dose is repeated every two or three hours as necessary until, if required, five doses have been injected. The stomach, always in the case of children and usually with adults, is emptied by lavage, but with the utmost gentleness. Sodium bicarbonate is not used.

I have seen patients in diabetic coma successfully treated in their own homes and by their own doctors. I have three impressions in regard to the home management of diabetic coma which perhaps are worth emphasizing.

The factor of time in treatment is of great importance. To treat coma successfully, it must be recognized early and treatment must be begun immediately. When a doctor gets a hurry call from one of his diabetic patients, he should answer the call as quickly as possible. If his patient is nauseated or vomiting, he should be on the lookout for coma.

Infections may not only cause a patient to develop coma, but also add a grave hazard to an already serious condition. The patient bordering on coma should be protected from acquiring infection in every possible way.

It is necessary for the doctor in charge to "camp" on every coma case. Unless the treatment is supervised carefully during the few days of critical illness, the result will not be satisfactory. Patients slip into and out of acidosis with extraordinary rapidity.

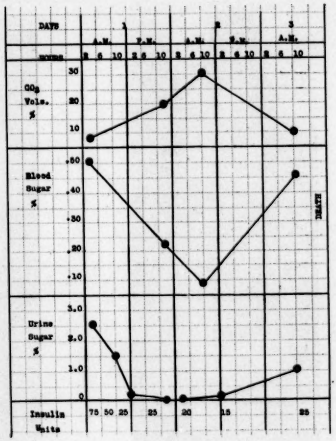


FIGURE 7. A Case of Diabetic Coma Treated Inadequately. A woman, 21 years old. Diabetic symptoms for a few weeks. In coma at entry to Hospital. Death three days later.

Figure 7 illustrates the changes occurring in the blood and urine, hour by hour, in the case of one of our failures. During the first twelve hours, one hundred and fifty units of insulin were injected and during this time, acidosis began to decrease, the blood-sugar to fall and glycosuria to disappear. During the second twelve hours, only twenty-five units of insulin were necessary to maintain proper progress. At two P. M. of the third twelve-hour period twenty more units were injected. The urine was sugar-free, and a few hours later the blood bicarbonate concentration had risen to thirty volumes per cent. while the blood-sugar reading was only .07%. No more insulin was given for twelve hours and then only fifteen units. Chemical analyses of blood and urine were not made. The next test, fifteen hours later, showed a return of acidosis, hyperglycemia and glycosuria. Death followed quickly. One cannot escape the impression that the patient would have had a better chance for recovery had the blood and urine been more carefully followed.

A doctor, therefore, who undertakes to steer a diabetic patient through coma must devote all his energies to the treatment of that particular case. He cannot do this over the telephone or without the laboratory facilities for testing the urine. If however, he recognizes diabetic coma early, gives large and repeated doses of insulin, an abundance of fluid, has a co-öperative nurse, follows the effect of his treatment by repeated urinalyses and carries out the general scheme of therapy outlined, I am convinced that the menace of diabetic coma will be materially lessened.

CONCLUSION

Diabetic coma is one of the most striking and potentially serious complications of diabetes. It still causes far too many deaths as shown by recent figures of the Metropolitan[7] Life Insurance Company. Up to April 15, 1929, coma was responsible for 433 deaths, or 41 per cent., of 1044 fatal diabetic cases.

Ten years ago, diabetic coma was almost always a fatal complication of diabetes and the cases surviving a severe attack of acidosis were likely to live for a comparatively short time thereafter. Even the best treatment of severe diabetics was unsatisfactory.

Insulin has made an extraordinary change in the prognosis of diabetes. Coma is no longer a fatal complication but is amenable to treatment. Comatose patients have a right to adequate treatment for many who have been in coma are now enjoying a happy and useful existence six or seven years after their initial attack and have even survived several subsequent attacks without great injury.

Diabetic coma should now be considered as a diabetic accident, preventable in most cases, having a good rather than a bad prognosis. It is no longer the natural culmination of the disease and is never a justifiable catastrophe.

The successful treatment of diabetic coma depends upon its early recognition. It must be attacked vigorously. It can be prevented in many instances by simple common sense measures. It can be treated successfully in the majority of cases by any well trained doctor. The treatment of coma consists essentially in the appropriate use of insulin, good nursing care and fluids.

The family doctor is under a definite obligation to his diabetic patients. He is the first to be called when any emergency arises. He has a great opportunity to establish the diagnosis of diabetic coma in its early stages and to prevent its development. Coma causes far too many diabetic deaths each year. The present high death-rate can be lowered if only doctors throughout the country will admit the practical importance of the problem and learn how to recognize and treat diabetic acidosis before it becomes dangerous.

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THE DIAGNOSIS OF NEPHROSIS*

BY ROBERT W. BUCK, M.D.†

THE term nephrosis does not carry with it the implication of any clear-cut clinical or pathological picture. It has been used to designate conditions which are dissimilar in etiology, clinical course and pathology, with resultant confusion and occasional impatience on the part of the clinician, who begins by attempting to learn what it is that is being talked about and how to recognize it, and who is apt to end with the impression that there is considerable doubt as to whether it exists as an entity in any case. The present brief review of the literature on the subject is presented with the report of a few cases, in the attempt to crystallize our present ideas in regard to the nature of the condition or conditions referred to as "nephrosis."

The word itself was proposed, innocently enough, on purely etymological grounds, by Friedrich Müller[1] in 1905 as a substitute for the clumsy terms "kidney of pregnancy", "amyloid kidney", "kidney of lead poisoning", etc., or instead of "nephritis" to designate those disease processes of the kidneys which are degenerative, or at least not clearly inflammatory in nature. Some accepted the idea and used the term; most did not, and it has never gained general acceptance.

In 1913 Fritz Munk[2], as the result of his studies in regard to the occurrence of doubly refractile lipoids in urine, described a form of degenerative kidney disease not previously recognized, which he called the kidney of syphilis. He observed a syndrome in syphilitic patients under his care, characterized by malaise, pallor, oliguria, edema of varying degree and marked

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albuminuria. This usually ran a self-limited course coincident with the syphilis and, unlike nephritis, ended in apparently complete recovery. The urine of these patients invariably contained droplets of lipoid material which had the property of transmitting polarized light in either plane. These lipoid droplets he found to be pathognomonic of lipoid degeneration of the proximal tubules of the kidney. The condition was non-inflammatory. It could occur in a pure form, or as a complication of a true nephritis. He might have termed it "subacute degenerative nephritis with edema", thereby avoiding the displeasure of those who will not accept the term "nephrosis". However, he simply referred to it as the kidney of syphilis.

Not long after this, in 1914, the work of Volhard and Fahr[3] appeared, in which some attempt was made to follow the suggestion of Friedrich Müller in regard to the classification of renal disease. They used the term nephrosis to include degenerative conditions due to chemical or bacterial toxins and metabolic disturbances, either directly or through "cloudy swelling". Under the head of nephritis, on the other hand, they included those conditions where, in the earliest stages, proliferative processes in the vascular supporting tissue dominate the picture. Accordingly, as this proliferation occurs primarily in the interstitium or the glomeruli, they distinguish interstitial or glomerulonephritis. Their third great division is the arteriosclerotic group of nephropathies. Munk, in view of the new popularity of the term nephrosis as a result of its use by Volhard and Fahr, adopted it and applied it to his "kidney of syphilis", which condition he now referred to as a lipoid nephrosis.

By thus modestly merging his new disease with the other nephroses, such as kidney of pregnancy and the kidney of corrosive sublimate poisoning, under a common terminology, Munk paved the way for confusion, although he still insisted that his lipoid nephrosis had a distinct entity which set it apart from all other nephroses. However, the confusion was more or less confined to the German school.

In this country it was not until 1917 that confusion was worse confounded by the appearance of a paper by Albert A. Epstein[4] on the "Causation of Edema in Chronic Parenchymatous Nephritis", followed shortly by another on "The Nature and Treatment of Chronic Parenchymatous Nephritis (Nephrosis)". Here the term is apparently used as a synonym for the old-fashioned condition which no one ever could diagnose in the living patient anyway, and which as a matter of fact has little pathological meaning, "chronic parenchymatous nephritis".

It was apparent that Epstein's work was purely clinical. The previous men had dealt with pathological entities. From Epstein's work one would not be forced to conclude that a new disease had been discovered. He described patients who had chronic nephritis with edema, and recorded his observation that some of them had a lowered albumin content with an unusually large amount of lipoid material in the blood serum. Richard Bright[5] had done as much when, in 1837, he said, in reviewing his labors of more than a decade, "If blood is drawn . . . the serum is often milky, and nice analysis will frequently detect a great deficiency of albumin".

Epstein's theory of the causation of edema in chronic nephritis has not stood the test of time any better than those it was intended to supplant, and although "Epstein's nephrosis" is still not a distinct clinical or pathological entity, his work, by arousing a great deal of interest in the subject in this country, has popularized the term, chiefly perhaps, because of his successful therapeutic use of a high protein diet in the cases which he has described.

At the present time, therefore, the term nephrosis means, according to the inclination of the user, either

1. Any degenerative disease process in the kidney, or
2. The lipoid degeneration described by Munk, or
3. The group of nephropathies accompanied clinically by edema and certain long recognized types of pathological change, not necessarily confined to any one portion of the kidney structure (Volhard and Fahr), or
4. A sort of composite of these three, which has a vague significance to a good many people, and which may or may not connote an alteration in the albumin globulin ratio in the blood serum, a lowered basal metabolism, the presence of lipoids in the blood or urine, marked albuminuria with low blood pressure and edema, etc.

It is thus necessary, in any discussion of nephrosis, to know which of the above four concepts is being considered. The following limitation of use of the term is suggested by the writer:

In regard to group (1), there would seem to be no valid objection to the acceptance of Müller's original suggestion, i.e., that the term nephrosis be understood to include all nephropathies that are distinctly non-inflammatory in nature, such as necrotic degeneration, fatty, hyaline or amyloid degeneration, albuminous degeneration ("cloudy swelling"), etc. Two obvious comments must occur to the unprejudiced observer, however. First, the classification is a pathological one. There is little possibility of determining clinically the precise nature of the pathological changes present, and if there were, the value of doing so is questionable. Second, what are we going to call inflammation? This question opens a field of controversy which has been rife in Germany, but which again is clinically of undemonstrated importance. Summarizing, we may say that "nephrosis" may be

accepted as a pathological term without significance in most instances.

On the other hand, in group (2), the lipoid nephrosis of Munk, we have a condition which is recognizable clinically, and which by reason of the nature of its onset, course and peculiar symptoms, deserves to be regarded as a separate disease entity. My own limited opportunity to compare pathological findings with clinical evidence of lipoid degeneration in the renal tubules does not enable me to present actual proof that the condition is diagnosable during life and demonstrable at post mortem, but I have certainly had the satisfaction of making the diagnosis and confirming it at autopsy. Murphy and Warfield[6] accept Munk's term, and in their very clear and valuable discussion of lipoid nephrosis report the case of one patient who was followed throughout the course of his disease. It is highly significant that this case, which conformed clinically in all respects to Munk's lipoid nephrosis, showed no acute or chronic inflammatory changes in the glomeruli of the kidneys. The capillary loops were everywhere normal. On the other hand, the proximal convoluted tubules revealed extensive and profound degenerative changes with doubly refractile lipoids in the cells and interstitial tissues. One such carefully followed case is of great value in proving the existence (which has been disputed) of the condition under discussion.

Groups (3) and (4) above noted serve, it seems to me, only to add confusion and should be discarded.

In the present paper we are dealing with lipoid nephrosis as defined by Munk, i.e., a definite symptom complex, the diagnostic feature of which is the presence of doubly refracting lipoid material in the urinary sediment.

The symptoms of this disease are as follows:

The onset is characterized subjectively by vague, general symptoms such as fatigue, loss of appetite, backache; objectively by slight pallor and perhaps some suggestion of puffiness of the features. After a varying period of time, the onset of definite edema brings the patient to his physician. This edema may be associated with shortness of breath and headache, but in the typical case, there is no objective evidence of cardiac disease or renal failure in the ordinary sense. With the onset of edema there is observed oliguria, and the urine shows an excessive amount of albumin (0.4 to 4.0%). It has a high specific gravity corresponding to the oliguria. The sediment shows many casts, epithelial cells, fat droplets, some leukocytes, and perhaps a few red blood cells. When examined with the polarizing microscope, the fatty elements are seen to be in large part doubly refractile.

The disease may progress no further and recovery may ensue. Often, however, the albuminuria persists for weeks and months. Often, too, in spite of apparent clinical recovery, marked edema may recur; the loss of appetite and headache are again complained of, and there may be somnolence to a degree that suggests uremia. Confounding the grave prognosis which this may elicit from the physician who considers that he is dealing with nephritis, the patient may again recover after having been in this pseudo-uremic condition for days or weeks.

Apparently there is no excessive load put upon the heart and circulatory system by these vagaries. The patient may have a normal blood pressure throughout. Owing to the lowered general resistance, complications due to secondary infection may occur. The pneumococcus would seem to be a favorite invader. Whether the disease may progress to a true nephrotic contracted kidney, and whether or how it may be complicated by the vascular or toxic changes of a true nephritis, are still matters of obscurity and controversy.

It seems certain that patients may recover completely from uncomplicated lipoid nephrosis, even though their symptoms are severe to a degree which would be almost certainly fatal if nephritic in origin. These are, no doubt, the cases in which Epstein has observed such striking results under high protein diet therapy and which others have described as having been rescued from imminent death by huge doses of thyroxin.

Lipoid degeneration is by no means always a separate entity. It not infrequently is met with in association with true nephritis, where it may, if unrecognized, confuse the clinical picture and lead to a more serious prognosis than is warranted.

The etiology of lipoid nephrosis is obscure. Apparently the state of nutrition of the patient plays some rôle. The condition seems to occur in so-called "anemic", debilitated persons. The starvation nephrosis reported by Russian observers may be significant in this connection. Chemical irritants play no part clinically, although lipoiduria has been produced in cats by first poisoning the kidneys with uranium and then feeding cholesterin[7]. The condition has not been reported as a complication of acute infection. It can occur as a manifestation of the toxemia of pregnancy. Chronic disease is the usual precedent. In tuberculosis it may be found associated with amyloid degeneration. Patients with malignant disease may show doubly refractile bodies in the urine without concomitant albuminuria or edema. According to Munk, the classical virus producing the picture is that of syphilis. Apart from the true "kidney of syphilis", pure lipoid nephrosis is rare, and more commonly than in any other associated conditions mentioned above it is found in association with or as a complication of a definite chronic glomerulonephritis, as the so-called "mixed type". Pathologically, the kidney of "pure" lipoid nephrosis is usually somewhat enlarged at the maximum stage of the illness. The color is usually pale grey to dirty

yellow. On cross section, the areas of lipoid degeneration are seen as fine, clay colored spots. Microscopically the cells of the proximal convoluted tubules show lipoid droplet degeneration. The cells of Henle's loop and those of the distal convoluted tubules show no change, nor do the glomeruli or interstitium.

The clinical pathology is fairly definite, but as yet not satisfactorily explained. The proper evaluation of these data has been made difficult by the confusion in terminology. Much of Epstein's work and that of other workers may have dealt with cases of chronic nephritis with edema which are not known to have been true lipoid nephrosis.

One of Epstein's first observations was that certain cases of nephritis with edema showed, together with marked albuminuria, a lowered total protein content in the blood serum, with a relative increase in globulin, so that the normal albumin ratio of 3/1 was reduced to 1/1 or even to a more marked degree. He attributed the edema to the fall in osmotic pressure of the plasma consequent upon its low protein content. The normal blood serum has more colloid (protein) than the tissue fluid, hence has greater osmotic pressure. Van Slyke et al[8] studied cases of chronic nephritis with edema which they assigned to a "nephrosis group", using as their criteria extreme edema, the presence of large serous effusions, scanty urine of high specific gravity containing much protein and many casts but no blood, the absence of anemia, increased blood pressure, retinal hemorrhages or exudate together with normal non-protein nitrogen in the blood. This group, of course, may or may not have included cases of lipoid nephrosis. The series included only five cases. They found in these five cases that the total plasma protein averaged less than five grams per 100 c.c., and that the albumin globulin ratio was less than one. They also felt that the greatest decrease in plasma protein did not correspond with the most marked edema and a decrease could occur without recognizable edema. They concluded that there is no direct relationship of cause and effect between low protein concentration in the plasma, and the presence or degree of edema, and likewise that when there is edema, the passage of protein into the tissue spaces is probably not the prime factor in producing a decreased concentration of protein in the blood. They suggest that possibly the body's most primitive form of protein production is the formation of globulin, and that in disease the organism tends to return to this primitive form. They also found that there was no satisfactory correlation between tubular fatty degeneration with edema and low plasma protein. They state that no hydremic plethora existed and that the two probable factors causing the decrease in plasma protein are (1) a disturbance in the mechanism of protein production dependent on the nature of the disease, and (2) the loss of large amounts of protein, especially albumin, in the urine.

These results are interesting but cannot be considered conclusive in regard to lipoid nephrosis inasmuch as we do not know whether the cases they studied had lipoid nephrosis or not.

Munk does not discuss the matter of reduced plasma protein, but he does stress the increase in the euglobulin fraction in the blood plasma and the hypercholesteremia. Epstein has also, from the first, mentioned the increase in blood cholesterol which occurs in his cases of chronic nephritis with edema. Munk believes that lipoid nephrosis is a constitutional disease affecting the colloids of the body fluids and cells, causing them to become more hydrophilic, so that water is retained by them in an abnormal way. He considers this to be the essential basis of the disease. He looks upon the condition as the result of a disturbance of the lipoid chemistry of the body. The lipoids occupy a position in the colloidal structure of the body fluids at the border line of the gels. They are contained in the euglobulin fraction and may be precipitated out by either the addition of electrolytes or by the electric current. Weill[9] says that the cloudiness of the serum and the transudate in lipoid nephrosis is caused by the precipitation of lipoids. These precipitated lipoids are denatured and act as foreign bodies in the blood and lymph and are excreted by the kidneys.

In explaining the edema which occurs in lipoid nephrosis, Munk recalls that the colloids of the blood serum may be divided, according to their physico-chemical relations to water, into hydrophile and hydrophobe groups. The albumin fraction belongs in the hydrophile group; the euglobulin fraction containing the lipoids belongs in the hydrophobe group. Normally the dispersity of these colloids is such that an optimal fluid exchange between serum and tissue is maintained. He states that the various protein bodies in the blood serum can be changed from one form to another according to the ionic concentration of the serum or by the electric current, i.e., albumin may become euglobulin. This change can occur as a result of nutritional conditions, fever, and the effect of bacterial toxins. His hypothesis is that in lipoid nephrosis there is a transformation of albumin to globulin (hydrophiles to hydrophobes) with a resultant disturbance of fluid exchange, the retention of water by the body, i.e., edema. He was able to give support to this theory by experimenting on dogs. He perfused dogs' kidneys with their own serum diluted 1:5 with Ringer's solution. The excreted urine showed only small amounts of albumin. Now, if an electric current of 1 to 2 volts was passed through the serum, the albumin changed into euglobulin. The euglobulin was precipitated out, and was excreted as a foreign body by the kidney so that there was a marked increase in the urinary protein.

He thus explains all the cardinal symptoms of lipoid nephrosis on the basis of an alteration in the body colloids consisting in a change of sols to gels, from hydrophiles to hydrophobes. The increase of hydrophobe colloid in the tissues causes oliguria and edema as a result of the attempt to maintain the optimal colloid structure of the body juices for fluid exchange. The precipitated proteins are excreted by the kidneys, giving rise to the symptom of albuminuria. Among these proteins which are excreted are the cholesterin esters which appear as doubly refractile lipoid bodies.

It follows then that there is a reasonable theoretical basis for confining the diagnostic term "lipoid nephrosis" to those cases where the typical lipoids are found in the urinary sediment.

The other clinico-pathological findings which have been noted in vaguely defined cases of so called nephrosis, such as lowered plasma protein, lowered basal metabolic rate, together with the various clinical findings, are of incidental but not diagnostic importance.

We believe from the evidence at hand that in order to make the diagnosis of lipoid nephrosis it is essential that we recognize the presence of the characteristic doubly refractile lipoid droplets (cholesterin esters) in the urinary sediment. To do so we must use the polarizing microscope. The ordinary microscope is easily converted into an instrument adapted for this purpose by fitting into the place of the Abbé condenser of the microscope a condenser containing a Nicol's prism, which serves as a polarizer. Another Nicol's prism is fitted into a loosely revolving cap which slips over the eye piece of the microscope and acts as an analyzer. Thus the light coming through the Nicol's prism below polarizes and illuminates the slide, passing up through the objective. Viewed thus, the field appears just as it would by ordinary light. When the analyzer is rotated, and the Nicol's prisms are crossed at right angles, the beam of polarized light is reflected off and the field appears dark. Now, if there are any bodies in the field which have the ability to rotate the plane of polarized light, they will be illuminated and stand out like bright stars in a black firmament. The lipoid material found in the urine of patients with lipoid degeneration in the kidneys has this property.*

In summary we may say that, confronted with a patient who presents the symptoms of pallor, fatigability, oliguria, edema and albuminuria, the diagnosis of lipoid nephrosis should suggest itself as a probability. If an examination of the urinary sediment shows the presence of doubly refractile droplets, the diagnosis is assured. We shall probably find that there is a diminished phthalein excretion, that there is an abnormal response to the dilution and concentration test of Volhard with a tendency to a fixation of gravity at a high level but, unless there is a coincident nephritis, there will be no evidence of nitrogen retention. We shall probably find that the blood cholesterol is considerably increased over the normal figure of 150 to 200 milligrams per 100 c.c. If the protein content of the blood plasma is studied, it will probably be found that this is low and that the albumin globulin ratio is reduced. We may find that the basal metabolic rate is low, partly owing to the fact that it is impossible accurately to calculate the normal rate if the body weight is increased by edema, but perhaps partly due to the nature of the disease itself. We shall probably find that the blood sedimentation rate is considerably increased. It is quite possible that in addition to these findings we shall discover that there is also a definite nephritis present, in which case we may find an elevated blood pressure, nitrogen retention, changes in the retinae, etc. In the latter case we should expect to find some evidence of cardiac enlargement. The purer the type of lipoid nephrosis, the fewer complicating factors will be discovered. Ordinarily we are not confronted with any difficulty in determining that the edema present is not cardiac, but if cardiac failure is present, the clarity of the picture is of course obscured.

The treatment of lipoid nephrosis does not as yet rest upon any firm theoretical foundation. The maintenance of the nutrition of the patient is probably more important than any rigid restriction of the diet. Prolonged rest in bed serves the purpose of conserving vitality and affording opportunity to the physician for careful study of the patient. The salt intake is restricted but not to an onerous degree. Patients should be treated symptomatically. Epstein, as is well known, has urged a high protein diet on the theoretical basis that this will restore the normal protein balance and increase the osmotic pressure of the blood. Although his theory is not sound, his patients have shown at times striking improvement. It must be remembered, however, that just as striking improvement has occurred in patients under observation who were not receiving a high protein diet. A sudden marked diuresis can occur as part of the course of the disease. Epstein has further reported improvement, in some cases which show a low metabolic rate, after the administration of thyroid. There have been reports in the literature of improvement in cases of chronic nephritis with edema (which, to be sure, may not have been lipoid nephrosis), after the removal of various foci of infection. Most of these therapeutic measures are purely empiric and have

*The appearance of the black cross in the center of the lipoid droplet is caused by the failure of rays passing through the droplet in certain directions to undergo rotation, because they are vibrating in such a relation to the optic axis of the crystal that they are transmitted without any change in their polarization. All such are cut out by the analyzer and form the black cross. A similar cross may be seen in examining starch granules by polarized light using the analyzer. They are larger and have a different appearance, however, from the lipoid droplets and further, they may be recognized as starch when viewed by the straight beam of light. Vegetable fibres, hair and crystals may all be easily distinguished from the lipoid bodies, although they all rotate polarized light.

not been controlled, so that their value is, to say the least, doubtful.

During the past six or eight months, during which I have examined the urinary sediment of all those patients whose urine showed moderate to large amounts of albumin, I have been forced to conclude that the occurrence of doubly refractile lipoid bodies is not common. I summarize a few typical cases showing lipoiduria. No attempt has been made to carefully study these as a group. However, where studies have been made, they have been generally consistent with the above given diagnostic characteristics of lipoid nephrosis. Most of these patients are still under observation.

CASE 1. M. L. C. ? Simple lipoid nephrosis, lipoiduria. A girl of 18, who, several months previously, first complained of transient slight edema of the hands, barely perceptible save in the morning. Her physician states that the urine had a specific gravity of 1.012 and showed a moderate amount of albumin, but nothing else of note. The sediment contained fine granular casts and occasional red and white blood cells. There was no past history of syphilis or chronic disease. Her blood pressure ranged between 130/80 and 140/90. The urine, when I examined it, contained 0.5% of albumin. The sediment was loaded with doubly refractile lipoid droplets and numerous hyaline casts with adherent lipoid droplets.

CASE 2. H. D. ? Subacute nephritis, lipoid nephrosis. A boy of 11, who complained of puffy eyelids of four months' duration. One month after the onset his legs and abdomen swelled. He was treated in one local hospital for acute nephritis and at this time he had many frontal headaches. He was later admitted to another hospital, where the same diagnosis was made. Urine examination showed a large amount of albumin and numerous granular casts in the sediment, which, when examined with the polarizing microscope, showed many doubly refractile lipoid droplets.

CASE 3. L. H. Toxemia of pregnancy, lipoid nephrosis. A primipara, age 22, 7½ months pregnant, who gave a history of swollen hands and feet, and headache. Eye grounds negative. Blood pressure 140/98. Phthalein excretion 40%. Non protein nitrogen 33 mgm. /100 c.c. Blood urea nitrogen 17 mgm. /100 c.c. Blood uric acid 5.1 mgm. /100 c.c. Blood sugar 101 mgm. /100 c.c. The specific gravity of the urine varied between 1.004 and 1.040. The urine contained very large amounts of albumin consistently. The sediment showed doubly refractile lipoid droplets.

CASE 4. E. C. Toxemia of pregnancy; lipoid nephrosis. A primipara, age 38, 6 months pregnant. Complained of slight edema of the ankles and an elevated blood pressure. Blood pressure at entry was 154/86 and rose to 188, at which time she was delivered. The urine showed large amounts of albumin consistently. The sediment contained doubly refractile droplets. Her hemoglobin was 60 to 65%. Red blood count 5,000,000. Phthalein excretion 60% in two hours. Non protein nitrogen 27 mgm. /100 c.c. Blood urea nitrogen 8.1 mgm. /100 c.c. Blood uric acid 3.3 mgm. /100 c.c. Blood sugar 83 mgm. /100 c.c.

CASE 5. E. J. Toxemia of pregnancy, lipoid nephrosis. A multipara of 31, 8½ months pregnant. Complained of rather sudden onset of marked edema. Her blood pressure, which had been 110, rose to 145/90. Her urine showed large amounts of albumin. The specific gravity ranged between 1.012 and 1.020. The sediment showed a few red and white blood cells and numerous doubly refractile droplets. Blood urea nitrogen 8 mgm. /100 c.c. Blood cholesterol 400 mgm. /100 c.c.

CASE 6. M. C. Syphilis; cardiac hypertrophy; ? cardiac failure; lipoid nephrosis. A woman of 46 years. Complained of edema, oliguria, fatigue and shortness of breath. Physical examination showed some edema of the eyelids and legs. The heart was enlarged as shown by X-ray examination and râles were heard at the bases of both lungs. Her blood pressure ranged between 154/90 and 170/90. With rest in bed and digitalis the lungs cleared, but the edema of the eyelids and legs did not disappear. The urine contained 0.5% of albumin. The sediment showed numerous casts, some red blood cells and abundant doubly refractile lipoid droplets. Phthalein excretion was 19% in two hours. Non protein nitrogen 13 mgm. /100 c.c. Blood urea nitrogen 19 mgm. /100 c.c. The total plasma protein was 4.8% (albumin 2.24%, globulin 2.56%). Blood cholesterol 187 mgm. /100 c.c. Blood sedimentation was over 30 mm. at the end of 1 hour. Basal metabolic rate minus 20. The dilution and concentration test showed a tendency to fixation of specific gravity between 1.017 and 1.020. There was likewise water retention, as shown by the fact that only 200 c.c. of urine was passed within four hours after the ingestion of 1000 c.c. of water. The Hinton blood test for syphilis was positive.

CASE 7. J. I. Pulmonary tuberculosis; chronic myocarditis; chronic nephritis with edema; amyloid disease; lipoid nephrosis. A man of 55, who complained of swelling of the legs and shortness of breath. The belly wall, thighs and legs were markedly edematous. X-ray of the chest showed slight enlargement of the heart and moderately advanced pulmonary tuberculosis. Guttadiaphot positive. The urine showed 0.5% of albumin. The sediment showed abundant doubly refractile lipoid droplets when viewed by polarized light. It was not possible to start kidney studies for some weeks after the patient was first seen. By this time his condition was grave and he was sent to the hospital, where he died. An autopsy confirmed the diagnosis of pulmonary tuberculosis, and the kidneys showed considerable diffuse nephritis with a good deal of amyloid change and extensive lipoid degeneration of the tubular epithelium. When frozen sections were stained with sudan iv and haematoxylin, characteristic lipoid degeneration was easily demonstrated. Examination of unstained sections under the polarizing microscope showed the presence of doubly refractile lipoid substances.

SUMMARY

Because of the confusion which exists in regard to the significance of the term nephrosis, it is suggested that its use be confined to the pathological designation of those nephropathies which are degenerative rather than inflammatory, in accordance with Müller's original suggestion.

The clinical use of the term should be limited to the diagnosis of lipoid nephrosis, inasmuch as this is the nephrosis of chief clinical importance which can be diagnosed with certainty. The demonstration of doubly refractile lipoids in the urinary sediment is essential for this diagnosis.

The symptomatology and clinical pathology of lipoid nephrosis are described, and theories concerning its etiology are discussed.

A few typical case histories are presented.

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CODMAN'S REGISTRATION OF BONE SARCOMA AND ITS BEARING UPON A STUDY OF MALIGNANCY ELSEWHERE

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I HAVE just finished reviewing a monograph on "Chronic Cystic Mastitis," written from the material of the pathological department of the University at Oslo, Norway, and published in 1928. The studies were made by Carl Semb, first assistant to Professor Francis Harbitz. It is a very careful clinical and pathological investigation of 144 cases of what they call *fibroadenomatosis* cystica mammae. The entire literature is reviewed and a number of very clear photomicrographs presented, which, however, are by far insufficient to picture this disease or its differentiation, microscopically, from early cancer. The conclusions of this investigation are practically the reverse of those of my own published in 1921, on some 350 cases of the same condition, and more recently on some 700 cases. Carl Semb identifies himself with those students who have come to the conclusion that chronic cystic mastitis is a precancerous lesion, and that it is wiser to remove the breast, even when there is no positive microscopic evidence of cancer at the time when the breast lump is explored.

Years ago, beginning in McCallum's laboratory in New York, and continuing for some years in laboratories in different cities, I submitted sections of some 60 breast tumors which had been diagnosed variously in my laboratory and followed for more than five years. There was no agreement, and some of the sections were examined by more than sixty pathologists in the different cities. The conclusion that the disease was malignant varied from twenty to eighty per cent. None of these patients have developed cancer in the breast, if it had not been removed at the time of the operation, nor did the glands show metastasis in the cases in which the complete operation had been performed. In this group of border-line breast lesions about which pathologists disagreed the only uniform characteristic was that *none* of the patients died from cancer of the breast; in fact, most of them are living today.

When I took this group out of cancer of the breast without metastasis to the axilla, the five-year cures were reduced from 85 to 70 per cent. In every clinic throughout the world that publishes the operative cures of cancer of the breast, there is always the possibility of this element of error. In my clinic this amounted to the difference between eighty-five and seventy per cent., or 15 per cent. It is important to note that we did not include among the possible malignant tumors all the border-line tumors. Chronic cystic mastitis should be subjected to the same searching investigation as Codman did with bone sarcoma. There is no doubt that Codman's registry excited great interest in this country and Canada, and the literature arising from this stimulation influenced the entire medical world to pay more attention to bone tumors. Codman requested, first, that every surgeon or pathologist who had the record of a patient who had lived or was living five years after an operation for bone tumor which was diagnosed as sarcoma, send the record to him and he would submit it to a committee of pathologists. I do not know the exact number of cases submitted, but the first report accepted only thirteen as verified sarcoma, which was a relatively small per cent. of the total number of cases registered. The largest number registered not accepted as sarcoma belonged to the giant-cell tumor group.

I am convinced that Codman's method will accomplish the most accurate results. Other committees are registering different types of malignant disease. Many, perhaps the majority, of them are accepting the pathological diagnosis of the individual or the clinic referring the case to the registrar. I would urge all these committees to return to the original requirements of Codman. The registry of bone sarcoma

*For record and address of author see "This Week's Issue," page 1008.

is being carried on by the American College of Surgeons under the direction of Dr. Crowell. The chairman of the pathological committee is Dr. Phemister of the University of Chicago. These verified cases can be studied by any member of the medical profession throughout the country.

The teaching of surgical microscopic pathology to medical students and postgraduate students should, as far as possible, be done with microscopic sections of tissue removed after at least a five-year interval has passed, so the student, looking at the section and being informed of the extent of the operation, may know the result.

In this country, a very large number of women have received and understood the message in regard to their breasts. The earlier women submit to a careful examination of the breast, the more frequently will chronic cystic mastitis be palpated as a single, multiple, or bilateral lesion. The examiner must learn to differentiate by palpation the "shotty", the "lumpy" breasts and those with dilated ducts beneath the nipple, and the definite multiple lumps in one or both breasts. For this group operation is not indicated, and the check of the correctness of the clinical diagnosis is the follow-up. Since 1920, the proportion of breast lesions in my clinic for which operation is not indicated has increased to sixty-five per cent. from one. My studies seem to show that these women run no more risk of cancer than any other group of women of the same age, followed the same number of years. In the group of cases of lumps in the breast in which operation is indicated, THE PER CENT. OF CANCER HAS FALLEN, in thirty years, from eighty to seventeen per cent.

The most common benign lesion of the breast which gives rise to a single palpable lesion, definite or indefinite, is chronic cystic mastitis. Its most frequent type is a blue-domed cyst; the next is a non-encapsulated area of chronic cystic mastitis. When there are submitted to a group of pathologists sections of breast tissue surrounding a blue-domed cyst or sections of a non-encapsulated area of the breast which has been removed, the diagnoses can be divided into three groups. In the first, the majority, if not all, of the pathological consultants will agree to the diagnosis of benignancy. Here there will be sections of fibroadenoma, intracanalicular myxoma, the simple adenomatous stage of chronic cystic mastitis, and the stage of ectasia or cyst formation. In the second group there will be a great difference of opinion—benign, suspicious of malignancy, malignant adenoma, adenocarcinoma, definitely cancer, or the pathologist may look upon it as a precancerous lesion and advise the removal of the breast to protect the patient against the future development of cancer. In the third group, there will not be a single difference of opinion among the well-trained pathologists. All will agree upon the diagnosis of carcinoma.

If group 2 is as benign as group 1, and if chronic cystic mastitis is not a precancerous lesion, it is a very essential thing today to bring together to study this problem, the pathologists responsible for the diagnosis of tissue in the operating room. When a doubtful lump in the breast is explored, and the lump is of short duration, as I have already stated, the chances of benignancy are at least fifty per cent., and the possibility of revealing an area of chronic cystic mastitis of the group 2 type is almost twenty per cent. In this group the picture is one of papillary cystadenoma, intracystic papilloma, small and large solid adenoma, often with destruction of the basement membrane.

In the past year I have been given an opportunity to examine more sections from clinically benign breast lesions about the diagnosis of which pathologists have disagreed than in any previous year. In addition, I have been surprised at the different interpretation of microscopic sections of distinctly encapsulated tumors of many years' duration. These women have had tumors of ten or more years' duration. They have recently learned that it is dangerous not to have an examination to find out whether the lump should be removed. The surgeon has palpated the lump, removed the lump with a zone of breast tissue, and then has been startled by being informed that is malignant. These encapsulated adenomas of the breast of years' duration are as different from the younger tumor in the young woman as the diffuse papillary cystadenomatous stage of chronic cystic mastitis is from the early adenomatous stage, or that of ectasia or cystic adenoma. In these old adenomas the stroma has compressed the parenchyma, destroyed the basement membrane, and you see epithelial cells loose in connective tissue, histologically suggesting malignancy.

I trust the committee of the American College of Surgeons, which has been and is still collecting the five-year cures of cancer of the breast, will insist upon the submission to them of the sections and that no case will be accepted unless the diagnosis of malignancy is accepted by the committee.

There is an opportunity to do for carcinoma of the breast what Codman has done for bone, and ultimately it must be done for the cervix and the uterus. We are entering the stage when our patients come to us with definite local lesions, the majority of which are not malignant. The final decision will rest upon the frozen section of a piece excised. Cancer is becoming more and more frequently a microscopic disease. The demand for frozen-section diagnosis in the operating room is beginning to be felt throughout the world, especially in this country. There must be found a way for training the young pathologist, and the pathologists already in active work, in the interpretation of these earlier

lesions, many of which are not cancer and never will be cancer; others of which are cancer in its precancerous stage, or the earliest period of malignancy. The time is not far distant when the microscope will be the instrument of precision which directs and controls the operative procedure.

I am impressed that one of the chief obstacles to this is the element of error, the difference of opinions given by equally good pathologists. Codman's registration of bone sarcoma has been an influence of tremendous value and has led and pointed to a way for the solution of the problem.

Undoubtedly many minds are working on the simpler diagnostic test for cancer. One would be a differential stain.*

*The distribution of information in regard to cancer through the influence of the American Society for the Control of Cancer and Codman's Registration of Bone Sarcoma has been coincidental with an increase of the five-year cures of bone sarcomas from less than four to more than thirty-five per cent. The earlier a bone lesion comes under observation the more difficult the diagnosis in the x-ray or in the biopsy.

I also wish to add the following figures in relation to lesions of the oral cavity, skin, breast and nodules to be felt beneath the skin. The relative proportion of these various lesions since 1925 as compared with the first decade up to 1900, is that in every locality—breast, skin, etc.—we see one hundred patients where before we saw only thirty-five. The incidence of cancer in these cases today is seventeen. In the early decade it was eighty. The cures of cancer in 1900 in these localities were less than ten. Today they are more than sixty. The per cent. of lesions that are not malignant and for which operation is not necessary has increased from less than one per cent. to more than sixty-five per cent. In the cases of local lesions which must be subjected to operation, the benign group has increased from twenty to fifty per cent., and the malignant group has decreased from eighty to fifty per cent. There seems no question that any activity which leads to publicity and gives the correct information to the people and to the medical profession, increases the cures of cancer. The complete control of cancer rests upon research.

REFERENCES TO THE INVESTIGATIONS OF BONE SARCOMA

Department of Pathology
Cornell University Medical College
First Avenue and 28th Street
New York

September 10, 1929.

Dr. J. C. Bloodgood,
Fairholt,
Burlington, Vermont.

My dear Dr. Bloodgood:

Dr. Codman's idea of an extensive country-wide study of bone sarcoma was the first successful demonstration of the great value of this method of investigation. International efforts of this sort have been previously attempted, especially in gastric ulcer, but they have met with no definite success. The success of the registry in bone sarcoma was due—first to the intrinsic merits of the project, and second and mainly, to the energy and foresight of Dr. Codman.

I consider the work of the registry as one of the outstanding American contributions to surgical pathology. As a method of study, it shows the necessity of very wide experience before a surgeon is capable of handling intelligently, cases of this disease, since the work of the Registry is impossible for any single individual to claim finality from his own experience in any important subject of this sort. The fact that the method has been adopted by several other Societies in other diseases shows that the lesson has been fully appreciated.

In foreign countries also, the work of the Registry has been valued very high, and will no doubt be followed in various places throughout the world.

Sincerely yours,

(Signed) JAMES EWING.

THURSDAY CLINICS OF THE BONE AND JOINT SERVICE (VI SURGICAL) BOSTON CITY HOSPITAL

TWO-STAGE PEDICLED GRAFT TO REPLACE UNSATISFACTORY SCAR

BY FREDERIC J. COTTON, M.D., F.A.C.S.,* AND RICHARD BERG, M.D.*

WE are reporting this case merely because it presents a rather interesting method of treating some of the more resistant types of ulcers about the weight-bearing or friction-bearing surfaces of the body. A male, aged 19, single, white, was admitted to the Boston City Hospital on January 2, 1929, with a diagnosis of chronic ulceration of the plantar surface of the right foot at the base of the fifth toe. His history in brief is as follows:

In May, 1928, the patient received a compound fracture, with dislocation of the right ankle, which finally healed and united with a slight outward rotation of the foot and about 20 degrees of plantar flexion. He had been up and about since, but in walking had developed an ulcer the size of a 50 cent piece at the base of the fifth toe. Local examination showed the patient in excellent health and physical condition, other than for the deformity which is described. The right leg showed three-quarters of an inch shortening with a mild equinus of 20 degrees and a slight amount of varus of the front of the foot. The foot showed about 30 degrees of outward rotation with the tibial shaft as an axis. This was undoubtedly produced by his fracture. The patient walked with the knee slightly flexed and with a step which necessitated throwing the weight in concentrated form over the head of the fifth metatarsal bone. At the base of the small toe there was a third degree circular ulcer with slightly infiltrated borders and a granular base the size of a half dollar. There was considerable tenderness around the area of the ulcer and apparently no definite progress toward repair. There was definite contraction of the tendon of Achilles.

Accordingly, the patient was admitted and boracic soaks and oil dressings applied to the foot and ulcerated area for a period of four days previous to the operation. In the meantime, the left leg was prepared for skin graft. On the 10th of January, a pedicle graft, with the pedicle from the anterior lateral aspect of the left midleg, was sutured into the freshened edges and the newly dissected bed of the ulcer on the right foot. The patient was put up in the "frog position" in a plaster spica in order to hold the limbs properly while the pedicle had a chance to heal. Four days later, January 14, 1929, a window was cut in the cast, and the graft was inspected. It showed a clean wound, and the edges were adhering well to the new ulcer wall. Eight days after operation, one half of the pedicle was severed; the graft continued to grow well and showed no evidence of gangrene. Three days later the entire pedicle was severed, and the remaining

*For record and address of author see "This Week's Issue," page 1008.

edge was sutured to the edges of the ulcer. Both wounds remained perfectly clean, and accordingly the patient was discharged the second of February. In order to correct the toe-drop, the tendon of Achilles was severed and stretched at the time of the operation, and a moderate square metal foot plate was fitted to the patient's foot. He reported back in a month, doing very nicely. The skin graft was entirely fused to the skin edges about the ulcer, and there was no evidence whatsoever of recurrence. The painful symptoms had subsided and the patient could go about without any aid and without any symptoms other than a slight limp, due partly to the outward rotation of his foot.

Our interest was attracted to this case because of the fact that numerous attempts in our out-patient department had been made to try and heal this persistent ulcer without avail. Accordingly the patient was referred to the Bone and Joint Service, and the possibilities of a graft were seen. These "full-thickness" grafts, in our opinion, are used to a far less extent than they should be, for as a method of healing over persistently denuded areas they are a treatment of proved value. In connection with this case, let me mention a similar graft done on a fireman who had a persistent cracking scar in the palm of his hand which absolutely resisted all methods of treatment. There was a definite fissure established, which was exceedingly tender and which prevented the full use of the hand. He was accordingly operated on, the scar tissue removed *in toto* from the palm of his hand and a pediele swung from the abdominal wall. The case did not do well, due to the fact that a rather large artery was by mistake included in the pedicle, and accordingly when the pedicle was severed, the graft sloughed out. It did, however, leave a very deep coating of fatty and subcutaneous tissue, which served as an excellent base for Reverdin grafts. There are numerous occasions where one could wisely use this pedicle graft, and it is to stimulate the interest in the recovering of denuded areas that we are reporting these minor cases in the hope that some one may thereby receive the benefits of such a simple and yet effective operation.

One of us reported certain cases in this class, years ago, in relation to the "lengthening of stumps"* and has operated in a lot of cases with intent to cover and slightly lengthen stumps instead of shortening them by re-amputation. Curiously, several of these have been heel-stumps, the result of that curious injury we used to call "escalator fracture".

Lately one of us has operated in two other cases; in one, successfully covering in the wide area of a crushed heel (of elevator, not escalator origin), using the other leg as source; and in the other, covering in a sensitive tight, ulcerated, thumb stump in a typical "stump-lengthening" operation, using belly skin and fat. Oddly enough, both these cases showed a phenomenon unusually seen in the writer's rather considerable series; namely, trophic ulceration of the graft. Obviously, such a graft has at the first, no nerve supply, and is open to trophic disturbance. Whatever trophic disturbance is, it is obviously due to nerve lesion or nerve lack.

In the heel case, there was primary healing, secondary ulceration at the edge, and tertiary healing with no evidence of infection, early or late. In the thumb case, primary healing was followed by a secondary plastic to fit the redundant edge, an operation for which no anesthetic, general or local, was needed. The edges did not cohere and an ulcer formed, which presently became beautifully circular and utterly inert. It healed under diluted boric wet dressings, almost exactly as sensation made its way into the anesthetic transplant.

There have been seen in these transplants, two or three times, odd pin-point sloughs, leaving minute ulcers that presently healed. It has been recognized that these transplants are oddly immune to infection—seemingly one may have blood circulation across the gap without lymphatic cross channels, and at all events one may have a sort of dull wound infection at the proximal edges, with an undisturbed graft.

The changes, apparently trophic and non-infective, to which the graft is subject, have not, we think, been worked out at all. However, interesting as they are, they are but temporary, never recurrent as far as we know, and in no way invalidate the great value of this method for covering defects and defective stumps.

*The Military Surgeon: 1919.

NON-UNION OF FRACTURES OF THE LONG BONES

BY FREDERIC J. COTTON, M.D., F.A.C.S.†

IT is not my intention, in discussing this interesting, often baffling, problem, to cover the field. Rather it is desired to bring forth some features observed in dealing with a good many cases. Therefore, I take the liberty of rambling through the subject, informally, in the hope that it may be of some value to some of you.

One may start with the statement that delayed union and non-union must be distinguished.

Delayed Union: Delayed union is a union between fragments of bone, which is not abnormal except for the *increased time element*. So far as the x-ray and the physical signs are concerned, it differs only insignificantly from the normal type of union.

Non-Union: In non-union, on the other hand, one sees a finished process, with ununited bone, and with the physical and x-ray signs which stamp it as an abnormal process. Non-union may show a pseudo-arthrosis which is caused by the interposition of soft tissue, either fibrous (fascial or tendinous) or muscular, or in some cases the periosteum. This may gradually cause a pseudo-bursa or joint to be formed between the ununited ends, or there may be only fibrous union without cavity formation. Once this stage has been established, union rarely, if ever, will take

†For record and address of author see "This Week's Issue," page 1008.

place without operative interference. In these cases, or in others without interposition, the callus in a non-union may be extremely abundant, and may, from an x-ray standpoint, resemble very closely the normal abundant callus in a healthy individual. If, however, non-union is present, by scrutinizing the edges of the fragments and the borders of the callus, one can usually see on either side a definite line of dense bone with a gap of fibrous tissue interposed between the sclerosed opposite surfaces; in between the bones are the fibrous tissues. The trabecular repair has failed to bridge the gap, and hence true cancellous bone is not thrown down. A sort of new joint without a joint cavity may be formed between the bone-ends, and, as the joint becomes functional, eburnated or hard cancellous bone is seen rounding off the ends of the fracture. Motion may remain trifling in extent; tenderness, instability and often increasing deformity are found. As a rule, bone will not grow through fibrous tissue. There are, however, cases which I have treated and seen, in which I am sure that solid cancellous bone has bridged a fibrous gap. This I believe is due to pressure and consequent new growth of bone laterally adjacent to the non-union area. I am firmly convinced, although I cannot prove the statement, that this type of fracture may come gradually to solid union by the progress, from a narrow bridge, of bone growth pressing and forcing fibrous tissue out of its way laterally. I believe this is what happened in a case I saw in a girl with a non-union after wiring of a transverse fracture high up in the tibia. I considered the case a definite one of non-union with the characteristic x-ray and physical signs described above. The patient, however, instead of submitting to re-operation, disappeared. I saw her again, ten years later, for another injury; to my surprise the old fracture was perfectly solid, with solid bone bridging the fibrous gap which had previously existed. I have seen one other like case. The rule, stands, however; non-union is non-union, and does not cure itself.

Some of the general factors which have to do with delayed union should be considered at this time. One of the foremost is syphilis. I am sure that syphilis, as a causative factor in non-union, is greatly over-estimated by the profession in general. There is no question but that it plays a very definite rôle, but there are hundreds of cases of normal union in definitely proved syphilitic patients. We see those every day in our hospital work on fractures, and it is only the rare cases that show any definite tendency toward non-union. Of course, in any such case, if delay in the healing-time takes place, we institute the accepted anti-syphilitic measures. As a hospital routine, cases with positive Wassermann or Kahn tests are treated, anyway, before letting them go.

Diabetes has been offered as another etiological factor but is of doubtful significance. In my experience, diabetes has played a very insignificant rôle in the problem of non-union.

Senility is of only relative importance. It is only in the extreme cases that non-union is to be laid to this cause. Of course, one sees a great deal of *delayed* union in the extremely old cases, and especially in cases of fracture of the neck of the femur. This cannot, however, be charged up to senility entirely; other important factors enter into the equation.

Paget's Disease: In this disease (osteitis deformans) delayed union is due to a local nutrition problem of bone. In this type of disease, the cortex of the bone is often very greatly thickened and of a much harder consistency than normal bone and hence softening and callus formation is necessarily delayed. However, union usually takes place after the normal softening process has set in. In the cases of this class, in which there is more absorption than sclerosis, we may run into trouble from sheer lack of lime-deposit. This, however, is very rare. With our newer knowledge of lime metabolism, we are better equipped to handle such cases.

Tabes is also a factor of delayed union. This is mainly due to the fact that sclerosed bone always requires considerably more time to soften in preparation for new bone formation. But fractures in tabetics do unite, in time. I can recall no case of real non-union.

There are many other factors in non-union. Perhaps one of the most prominent is the presence of infection. This plays a very important rôle, especially in the compound fracture, where one is dealing with a low grade of osteomyelitis and with soft tissue inflammation. The presence of infection, of course, delays the onset of bone formation. It is not necessary to discuss the question of infection in its numerous ramifications, for this feature, hardly to be formulated, is only too well known to all of us.

Some of the Local Mechanical Causes of Non-Union. The interposition of any soft tissue, usually fascia, or muscle, will, of course, constitute a definite mechanical hindrance to bone formation, though not necessarily an insuperable one.

Early mobilization is at times the mechanical cause of non-union, due to the fact that there is always a tendency to prevent new bone formation where edges of bone are constantly rubbing together. Probably, as cases run, this is a very important factor—imperfect fixation.

Lately the question of the lime balance in the body is a mooted factor. In spite of tests of the blood calcium, which show in most cases a normal blood calcium content, there is often obviously a dis-balance in the lime metabolism, a lack of the normal *deposition* of *lime salts where needed.* There is a definite lack of ability to lay down calcium at the site of callus formation, and yet the blood calcium findings will offer you no information. However, by the

administration of vitamins, especially D, best given in combination with cod-liver oil, and of irradiated vegetables* and parathyroid extracts, there does seem to be a beneficial action so that calcium is then laid down. Care must be taken in the administration of parathyroid extract alone, as it is a rather dangerous drug in some cases. Under its influence lime and phosphorous balance is preserved in the blood stream, but apparently at the expense of *any* source of lime, including bone! In this type of case the biological chemist is of great help to the bone surgeon. Better yet is the clinician accustomed to handling rachitis and scurvy in children.

Treatment: In general, the treatment of non-union simmers down to the non-operative and the operative procedures.

Non-Operative: In the non-operative treatment of non-union or delayed union, the surgeon may determine, insofar as possible, the etiological factor or factors, and attempt to remove them if possible. There are other adjuncts of definite value,—for example, diathermy, baking and massage, and the various physiotherapeutic agents, including the lights. One must strive to build up the patient's general resistance by the administration of vitamins, extracts, and substances of known value. Perhaps the most outstanding agent in this group is cod-liver oil. Remarkable results have sometimes been obtained by the administration of this simple substance alone. If infection is present, it must be thoroughly eradicated by proper procedure. If the fracture is showing a delayed or non-union due to *mechanical* incongruities, these must be remedied as soon as possible in order to prevent fibrosis of the ends of the bone. If the mechanical appliances are inadequate, some other method should be applied, or operative treatment instituted. Fragments which are widely separated tend toward non-union; therefore we should strive toward something close to the anatomical reduction of our fractures.

Operative Treatment: This consists mainly in bone grafts, and the different types of internal fixation, with freshening of the fragment ends.

It is only necessary to mention briefly the types of grafts which are commonly used for non-union of fractures. The first of these is:

No. 1 Sliding bone graft, which was originated in Italy† and which has since found favor throughout the medical world; it consists simply in sliding a graft from the adjacent bone, bridging the gap between the fracture ends. This works fairly well, and is comparatively easy to do.

No. 2 The Albee graft, which is the mass inlay graft, is very useful in certain types of fracture. This is very commonly used and is practical enough so that it can be employed with relatively good result.†

No. 3 The Magnuson Technique. This is not really a graft but an ivory inlay; the technique in this type of operation is very meticulous; it has shown good results for Magnuson, but due to the fact that it has not been used largely in other men's hands, one cannot report extensively on its general merit. The inlays are incorporated into the living bone, and the results seem excellent.

No. 4 The Intramedullary Graft. This is not very good because if the graft is large enough, it destroys far too much of the medullary cavity. One cannot set in an intramedullary graft without damaging the tissues of the canal for some distance above and below. A small autogenous graft, however, is all right in that it does not destroy so much of the cavity; but it does not fix the fracture as firmly as the larger graft. In my cases, I have had somewhat delayed union in all, but only one recurring non-union. Ryerson has been the advocate of this method, using beef bone inserts. I have tried this method in a series of cases with only one failure, but with much delay. I do not like it!

No. 5 The Osteoperiosteal Graft. Personally, I cannot see the special value of this type of graft over the plain bone graft. I believe it is just as well to freshen the periosteum about the edges of the bone, as to bother with periosteal grafts. A few years ago in conjunction with Dr. Halsey Loder,* I did some work on non-union in cats and rabbits to see what happened to endosteal bone in the new bone formation. We found that the growth of endosteal bone is very prompt indeed. Bone formation occurs by the laying on of the new bone on the existing trabeculae by osteoblasts. Calcification is quite rapid, bone growth is very rapid, and no cartilage is thrown down, as in the other type of bone formation where cartilage is gradually calcified. This work was done within the joint cavities of the animals in order to remove the possibility of soft tissue and periosteal interference. The net conclusion is that endosteal bone, under favorable conditions, unites by a sort of first intention, and that such repair is far more to the point than anything produced by repair from imperfectly approximated periosteal callus.

No. 6 The Compression Type of "Chip" Bone Graft. Working on the principle described briefly above, I have found that if one attacks a non-union by concaving the ends of the bone by means of a curette or chisel until he comes to definite normal

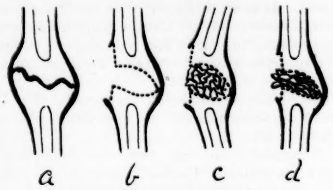
*We have done nothing with the irradiated ergosterol "acterol", as yet. It seems efficient, but too much like the super-efficient "TNT". When more is known, it will probably be very useful.

†Codavilla.

†Not uncommonly, we find union at one end and not at the other, of the graft, which isn't good.

*Cotton and Loder—Surgery, Gynecology and Obstetrics. Chicago: 1913, 16:701-703. "The Fate of Bone Grafts."

bone, not necessarily opening the medulla, then bending the bone ends outward so as to have an open cavity with the widest opening outward, leaving a layer of fibrous tissue or periosteum on the opposite side of the fracture to act as a hinge, one can pack very firmly small fragments of cortical, or, preferably, cancellous portion of the bone, carefully denuded of all periosteum, into this concave cavity. The fragments are then



TECHNIQUE IN DIAGRAM

a. Old non-union with thick periosteum, and with heaped up new bone at the sides; between the ends, a layer of fibrous tissue, often of no great thickness, and in incredibly irregular planes.

b. The fibrous tissue interval is dug out, and a cavity somewhat cup-formed to either side is shaped, going back beyond fibrosed bone to bone of good texture with free blood supply. Usually, and preferably, the marrow cavity is not opened. The thick fibrous tissues on the back side of the shaft are preserved for use as a "strap-fulcrum", as will appear.

c. The shaft is bent back to open up the cavity. The cavity is packed full of small "chip" grafts of cancellous bone, *without periosteum.*

d. The bone is brought back into a straight line; the strap of fibrous tissue acts as fulcrum; the mass of grafts is crushed into close contact, and so holds as long as the limb is kept straight, in apparatus of whatever sort.

jammed back into normal alignment, thereby further compressing the tiny grafts into this cavity, and bringing them into forceful contact with the roughened edges. The posterior untouched surface now acts as a strap fulcrum to hold the edges together, and the external fixation maintains the pressure. The periosteum or callus is then allowed to fold back over the wound, and the wound is closed in layers. I have obtained scores of excellent results with this type of operation, and intend to publish more fully the technique and results of a large series of cases at a later date. The detailed technique has been published.*

An important point is the convalescent care. Diathermy helps much. Early function and weight bearing in rigid convalescent apparatus are of first importance.

This in general covers the field of operative and non-operative treatment in non-union. Much more can be written on each of the details of this subject; these are the salient features.

*Journal of Bone and Joint Surgery, 10:94-95, Jan., 1928.

RAYNAUD'S DISEASE AND THE TRAUMA OF OCCUPATION

BY F. J. COTTON, M.D., F.A.C.S.,† AND R. BERG, M.D.†

IN May, 1929, there was referred to me a patient, aged 39, of Slavic racial antecedents, afflicted with trouble in the thumb and index fingers of the right hand. It seemed that her only mode of self-expression in this world for some years had been a position which necessitated her putting pins in pleats and shirts for a laundry concern. Without previous trouble of any sort she had noted, two weeks before, pain and coldness in the tips of the thumb and index fingers (right) with which her work was done. There was a slight involvement of the tip of the second finger also. There was not only pain but marked soreness to touch, coldness and change of color. As treatment by her doctor had proven of no avail and the condition was slowly growing worse, it had been arranged, through the insurance company covering the laundry people, to have a consultant see her; consequently she came to my office.

She proved to be a sturdy, middle-aged Slav of dark type, seemingly thoroughly normal in mind and body, not obviously neurotic and not the complaining sort.

Local examination showed the tip of the right thumb and of the index finger to be of a dull blue, very cold, very evidently shrunken in bulk, and very tender to even moderate pressure. The skin of the anterior surfaces of the thumb and finger tips and also in some measure the sides were roughened as one sees in seamstresses' fingers with the thousand stabs of their occupation, but nowhere was there any sign of actual wound, or sign of sepsis. The flesh was, as said, very definitely shrunken, the skin gray-blue, the nails almost cadaveric with hardly any return of circulation after pressure. The tenderness was very definite. All signs were confined to the terminal joints. The second finger showed roughening from her trade, and she said it had been tender and cold a few days before but was better now. There was no tenderness of her hand or arm, no epitrochlear or axillary gland enlargement. Dr. E. E. Bowen, of East Boston, who had seen her previously, confirmed, over the 'phone, her story, that she was growing slowly worse.

There seemed no shadow of question that we were dealing with a condition of vessel spasm. Confirmatory, for what it was worth, the right radial pulse was distinctly less in volume and harder in texture than the left, though the hand in general was only slightly, if any, cooler. Whether one called it Raynaud's disease seemed of no great moment, nor the apparent connection with habitual occupational trauma. The important fact was that we had an impending gangrene facing us, and the only chance seemed to be immediate recourse to foreign protein "insult" to produce dilatation. I didn't even wait to try out the relaxation following tourniquet application (Cushing's plan), as it seemed to me we had very little vital margin locally. She was accordingly sent directly to the hospital, and received that evening an intravenous injection of typhoid vaccine of 25 million killed bacteria. There was the usual delayed reaction.

The injection was given at 5 P. M. on May 21 and at 11 P. M. she had a definite chill; her temperature at the time of chill was 102°, the pulse had increased from 80 to 100, respiration remained normal. The

†For record and address of author see "This Week's Issue."
page 1008.

next morning when I saw her the picture was entirely changed. The fingers were almost normal in color and in capillary circulation. Oddly enough, the right radial pulse, previously thin, had become the stronger of the two. There was still, however, some tenderness of the finger tips to pressure. This tenderness persisted for four days, after which it entirely disappeared.

Two days later the second injection was given, for good luck rather than for symptoms. Thirty-five million bacteria were given this time, and, oddly enough, produced no chill, no break in the temperature line of the chart, the only reaction being a rise in pulse rate from 80 to 120. Nor did the hand, then already practically normal save for the roughened finger tips, show any change.

A few days later the patient was discharged apparently entirely cured. Ten days after the second injection the patient reported to my office perfectly all right as to the right hand; but she informed me that, the day before, the tip of the left thumb, previously perfectly normal, had become painful and bluish in color, for a period of a few hours. When I examined her she showed nothing in this digit. The right radial pulse was still a bit fuller than the left; otherwise she was apparently normal except for the relatively permanent skin or subcutaneous skin changes due to her work. While she was in the hospital, Dr. Richard F. Berg, Resident Surgeon, checking on the details and notes, added the following information:

On entrance to the ward, the patient was given the routine examination and laboratory tests. The urine and blood examinations showed no abnormal aspects. The white blood count was 8400 with a normal differential. The red blood count was 4,200,000, with 85% hemoglobin. Wassermann negative, Kahn negative, spinal fluid not done. Blood pressure on entrance was 125/80.

The patient was given the diluted typhoid vaccine, with approximately 25 million bacteria, intravenously in the unaffected arm, with no noticeable effect for 6 hours, following which, the usual chill and rise in temperature in pulse, characteristic of a protein reaction, ensued. The symptoms disappeared by the following morning and the marked improvement of the finger was noted, the index tip being the only remaining tender area. The patient had external heat in the form of a hot-water-bag applied to the hand, in conjunction with the vaccine therapy. When the temperature was normal (two days later), the next injection of 35 million bacteria was given intravenously with no response in constitutional signs other than an increase of the pulse rate from 80 to 120.

From this time on, the tenderness in the fingers rapidly subsided. As was previously stated, the bluish discoloration disappeared following the first injection. The radial pulse on the affected side was unbelievably increased, following the first injection, from a thready, hard pulse to a full, bounding one. The skin over the fingers had the glistening, worn aspect of the patient who uses the tips of the fingers a great deal; it felt as though deeply calloused or fibrosed, and on entrance the finger tips showed a definite shrinking in bulk giving a definite tapering off aspect. The patient was kept in the hospital for a period of 7 days following the second injection, merely to convince ourselves that the condition was really as greatly improved as it appeared to be. Accordingly, she was discharged to report to Dr. Cotton's office at the end of the 11th day.

Dr. Stanley Cobb of the Neurological Service saw her for us while she was in the hospital. His report is as follows:

"Scar of old thyroid operation with history of goitre. Right hand with first and second fingers showing trophic changes, pale, blue and tender, with history of an attack in the left second finger previously. Heterochromic iridis. Unequal pulses of the right and left radials; deep reflexes exaggerated with short clonus. Vasomotor reflexes exaggerated.

"*Impression.* A most interesting case of Raynaud's disease with an old thyroid tumor and now various signs of sympathetic instability.

"*Treatment.*

"1. Keep on with vaccine shock.

"2. If this fails, try Adson's operation on the stellate ganglion.

"STANLEY COBB."

Résumé. Fortunately, the case is an industrial one and therefore subject to follow-up, often impossible in cases of the drift of hospital jetsam. It is to be hoped that careful checking and administration of foreign proteins as needed may control the situation without the necessity of any sympathetic surgery such as Adson's ramectomy, or the like.

The point, in recording the case, is that Raynaud's disease should be approximately symmetrical, and that an association of the onset of Raynaud's disease and the local trauma seems, judging by all that I have heard and all my colleagues can tell me, entirely unknown. As the case first presented itself the connection of occupation with the trouble seemed inescapable. Dr. Cobb's examination showed clearly a sympathetic disturbance, or lack of balance in the background, in the sympathetic nervous system, and the later unmotivated involvement in the unused left thumb made it clear that we were dealing with an instance of Raynaud's disease, real enough, typical enough, not traumatic, or occupational at all, but existing in the "larvate" stage. That the first expression of this phenomenon should have been localized by occupational trauma is odd enough. All in all, the case offers a very interesting study in the multiplicity of vasomotor and sympathetic phenomena.

OSTEOMYELITIS — WITH SPECIAL REFERENCE TO TREATMENT

BY FREDERIC J. COTTON, M.D., F.A.C.S.*

THIS afternoon, let us take up the treatment of acute and chronic cases of osteomyelitis, and stress particularly the types of treatment which are found to be most satisfactory in practice. One does not see the text-book picture of osteomyelitis often, these days. Previously, one would pick up a case with a typical history, such as that of a small boy going swimming one day, slightly injuring his ankle or leg, or getting chilled; this being followed, within a day or so, by a sudden chill, high temperature, and the clinical signs of an acute osteomyelitis of the "typhoid" type. Such a case offers few obstacles to a diagnosis, if one thinks of the possibility, but as above said, we do not very often see such clean-cut clinical pictures these days. The "local M.D." has grown wiser.

Acute Osteomyelitis. It is not uncommon to see an osteomyelitis with a slow onset, lasting a week or so, which finally shows all the clinical

*For record and address of author see "This Week's Issue," page 1008.

signs of localized bone disease. These are the hard cases to diagnose, and these are the ones with which you will be confronted in your daily practice of medicine. The fulminating type comes on with a very acute onset, producing a tremendous toxemia or a septicemia over which the body has little control. On the other hand, one will see the border-line case, the slowly progressive type which usually gets better. This type allows one to make a satisfactory diagnosis —oftentimes early enough to prevent any serious bone necrosis.

With these principles well in mind, one must be very careful in examining a case of questionable character. If you see a case with any pain in a limb, especially the lower limbs, think of osteomyelitis. There may be no swelling, it may not be tender to touch superficially, but it will always be tender to direct tapping on the shaft of the bone. This is a point of great importance in differential diagnosis, where one must consider and rule out the possibility of a superficial cellulitis or an infection which extends down to, but not including, the bone. I do not remember having seen a proved case of osteomyelitis in which this sign was absent. The hard cases to diagnose and differentiate are those with a joint infection. In these cases, the diagnosis becomes very difficult, as the type of symptoms may be referable to the joint entirely and the osteomyelitis which is co-existent will often be overlooked until drainage has been established after bone necrosis. It is a sad thing that the x-ray is so useless in the early cases of osteomyelitis. Good x-ray men will tell you that the x-ray plate is of very little value within the first twenty-four to thirty-six hours of the disease.* There are instances, however, in which the x-ray pictures will show a cloudy trabecular structure: in these cases the x-ray plates are of some value.

In differentiating acute osteomyelitis, the most common thing to confuse a diagnostician is erysipelas, which gives a *superficial* inflammatory process, and which, in many cases, may be confusing. There is, however, a line of surface demarcation not present in osteomyelitis. Another thing which often confuses is a superficial cellulitis. In this the systemic reaction is less marked, the bone tenderness less specific. In infants, the nutritional diseases, such as scurvy and rickets, may temporarily confuse one, but upon observation these cases generally clear up with proper treatment.

Chronic Osteomyelitis: This is usually readily diagnosed by the existence of a sinus, either spontaneous or from previous operation on an acute case. The x-ray usually clears up the question of diagnosis in these cases. The treatment of this condition will be taken up with that of acute osteomyelitis.

*Dr. Paul Butler, radiologist, deserves great credit for his open declaration, often repeated, that the x-ray is *valueless* in the diagnosis of *acute* osteomyelitis.

Treatment: There have been several theories advanced as to the proper treatment of acute osteomyelitis, but each case presents an individual picture and requires technique which is modelled after its type or character. It is foolish to lay down any stringent law as to the right and wrong way of treating any acute case, but certain principles are accepted as facts, and upon this, one may base a line of treatment. The first principle is that of *immediate* and *adequate drainage.* For this one drills a few holes in the cortex, and then, pus once reached, by means of a small rongeur, a trough is channeled into the medullary canal, and to the site of inflammation. In this way, very little shock is experienced by the patient. This is better than subjecting an acute case to the trauma of chiselling. Very mild and early cases may clear up with simply the relief of pressure by a few drill-holes, but as a rule it is better to "play safe," and channel the cortex for adequate drainage.

The second principle is the application of suitable dressing. Since Orr has advanced his grease-dressing theory of treatment, we have been awakened to some of the facts which we had regarded rather lightly in the past few years. I, personally, used to use heavy grease dressings with very excellent results. In some cases I even used a bone wax, providing my field had been thoroughly sterilized by carbolic acid applications. This, however, is not for acute cases, useful as it is in chronic cases where the disease is fairly well localized. There is no doubt that grease gives a better protection to bone and to the soft tissues, preventing air-drying, and at the same time allowing adequate drainage, if not packed too tightly. I firmly believe that we get by with less bone-necrosis, also, with this method. If however, you do have extensive destruction of the bone before you operate, there will be a definite sequestration. On the other hand, early cases may get by without any sequestration. After you have operated, there is nothing further to do than to provide adequate drainage until the sequestrum forms.

Some men have advocated the "Ideal operation". This, in principle, consists of waiting until you get a definite beginning, paper-thin involucrum formed, as shown by the x-ray, then going in and taking out the sequestrum at the same time lifting the periosteum and later collapsing it, so that the two *inner* surfaces of the periosteum are in contact. This will be the basis for new bone growth. Periosteum, in these cases, generally shows a fairly good new bone formation. In operating, it is advisable to remove the whole segment of diseased bone and allow the periosteum to collapse completely. Thus you have a potential shell remaining with the new bone growth progressing on the inside. As a rule, if the operation is properly done, good bone will grow back, but occasionally one must re-operate and refreshen the edges. Some-

times periosteum is partly destroyed, and one gets the reformation of the shaft perfect *except* for a half-inch or an inch. That is why I do not do it this way, pretty as the way is when it works.

Ordinarily, one waits longer, until some *solid involucrum* is present. One may clear out a sequestrum earlier, if small; if large, you must wait until the sequestrum is definitely formed and demarcated, and new bone present in mass. An involucrum will heap up around most of the bone, forming an outer shell or coat. In these cases the sequestrum is already separated, and an infected cavity exists, inside the involucrum and surrounding the sequestrum. In such a case, it is not advisable to just channel away enough bone to clean out the sequestrum, but a great deal of the involucrum, should be thrown away, in fact, enough of it so that good solid bone is reached, in such form as to leave no deep pockets. This often necessitates removing one-half to three-quarters of the involucrum shell, and leaving only a *portion of the shaft to form the basis of new bone.* Of course, in such a case one must be very careful of a fracture, but in case this does occur*, there is generally very little trouble in securing good union, even in spite of an existing infection.† Even in the late cases, where considerable destruction has gone on, there will be sufficient regenerative power to grow new bone by this method.

In persistent cases with sinuses, it is often necessary to lay the bone wide open and to use very radical methods. In general *the results obtained are in direct proportion to the radicalism of the treatment* instituted. You cannot thoroughly disinfect in all these cases, but if you can thoroughly carbolize the wound**, you will generally get a complete disinfection, and bone will soon regenerate. If only partial disinfection is secured, it will go along fairly well, but the results will be longer in appearing. Where it is complete, one may often use bone wax††, and the wax will gradually be extruded by the newly growing bone and granulation tissue. Included wax seems to be absorbed slowly.‡ Generally speaking, if sterilization is a bit doubtful, use Orr's grease rather than wax, which *may* occlude drainage.

Orr has published his story.*** He has waked us up to the fact that rest is important, and that one gains by using grease dressings rather than the foolishly frequent dry or wet dressings we were brought up on. As I said before, I used grease years ago; bone wax as well. It does decrease and prevent the amount of sequestrum formed; no doubt about it. It also does away with the dead spaces in the tissues which tend toward infection and reinfection; it produces a local hyperemia, and prevents chilling. Do not use wax unless you think the wound is fairly well disinfected. Pack in your bulk vaseline, and then use gauze, loosely incorporated with the vaseline. Dr. Orr leaves the dressing until the smell becomes almost intolerable. However, it does not seem necessary to go that far with the treatment. We usually change our dressings about the tenth day, and by that time the primary result has generally been obtained. Granulation tissue is usually quite abundant. In this way the amount of sequestrum is reduced and secondary operation is less often required. Osteomyelitis requires great skill in treatment. One must study each case individually and attempt to carry out the principles which are best suited to each one.

Summary: We may then summarize our treatment of osteomyelitis in this way:

First: Early drainage in the acute cases; adequate drainage in the chronic cases.

Secondly: Proper after-treatment, which includes the dressings whether they be of grease or one of the other accepted types.

Thirdly: The radical operation of the chronic massive cases. The radical operation shows fine results, but it is a very serious one and should be done rapidly and thoroughly, due to the fact that there is usually a great deal of hemorrhage and shock. Frequent x-ray check-ups help us in determining the results in our early cases.

Prognosis: The prognosis, as far as immediate results are concerned, is always questionable, though usually good. As to life, it is very good, except in the case of osteomyelitis of the spine, pelvis, skull and hip joints, in which cases the reaction is very severe. The mortality is very low in infection of the long bones and the number of cases which can be cured is very high. There may be, however, recurrences, but these are usually a recurrence of sepsis in the scar rather than in the bone itself. Rarely, there may be a massive recurrence in the shaft. Spontaneous fractures are also rare, but in case they do occur, generally heal up fairly well. Recurrences often may be resistant, due to a sclerosis of the bone, due to poor nutrition, and these cases may never clear up. These cases, however, are in adults or in very old people. Fortunately, in the children, the prognosis is usually very good.

Drainage may be the only need. Removal of a local sequestrum later, if it is small, may suffice. Removal of sequestra, and of much of the violently growing involucrum, gives a clean field for disinfection and for later growth to something near normal.

The best results seem to be from

(a) Early, *complete* drainage of bone as well as abscesses.

*As it has occurred in three cases of mine, in the series.

†Oddly, there seems to be no re-infection at the ends, in no case any new sequestrum.

**Crude carbolic, 1-2 minutes; abundant alcohol to follow.

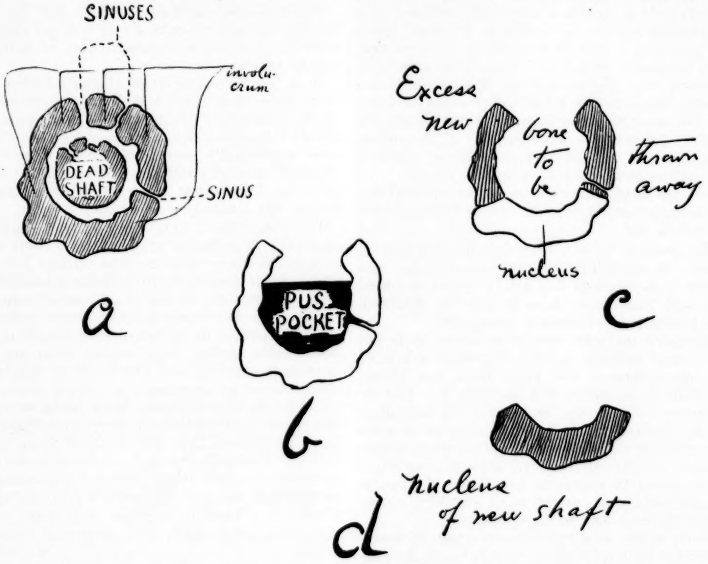
††Lukens' bone wax, on Horsley's formula. It is important to have an "animal" wax, not a paraffin base.

‡Case P.— S.— A mass of wax set in the tibial head five years ago has reduced as shown by the skiagraph to about three-eighths its bulk. The case was a large, clean, bone cyst.

***H. Winnett Orr: Osteo-myelitis and Compound Fractures. Mosby Co., St. Louis, 1929.

(b) Clean-up of partial necroses, as soon as the x-ray shows delimitation and beginning separation of sequestra, with either
(1) effective sterilization of the resultant cavity, and bone-wax filling in the cleaner cases,
or, (2) such wide-open clean-up as may be, and such sterilization as is possible; then dressing with grease packing.

(c) "Ideal" operation in a few picked cases of "total" necrosis of the shaft.



But, in other "total" cases, there is another way,—namely, (d).

(d) Operation, sufficient time after for growth of a decent involucrum*; operation consisting of removal of the sequestrum and of a most radical removal of most of the involucrum, sterilization, and, then, vaseline dressing.

(e) In chronic cases, go forward either as in (d), or in the presence of multiple sinuses, with a rather radical removal of bone, with curettage of sinuses, with such disinfection as may be practicable, and then with either Dakin treatment, meticulously carried out, or Orr's grease dressing, with immobilization.

So handled, osteomyelitis means a minimal count of deaths from sepsis, a reasonable number of very prompt cures *without secondary operation, and a majority of prompt and permanent* results where secondary operation is necessary.

There remain a few cases, calling for repeated clean-up operations; a few chronic cases that cannot be cleaned up, whether because of extensive sinuses in infected soft parts, or because of eburnated bone at the bottom of sinuses, particularly in chronic cases in elderly people; a few bone recurrences, maybe after years; many recurrent abscesses, superficial in the bone, or more often in scar tissue only, mostly trivial.

We have come to a point, I think, where we may regard our treatment of osteomyelitis as rational, as reasonably stabilized and reasonably successful.

*Eight weeks on the average.

"FENDER FRACTURE" OF THE TIBIA AT THE KNEE

BY F. J. COTTON, M.D., F.A.C.S.,† AND RICHARD BERG, M.D.†

THE automobile in contact with the jay-walking citizen has brought a previously rather rare fracture type to the fore.* This is the fracture of the outer side of the tibial head, pro-

†For record and address of author see "This Week's Issue," page 1008.

*Just as pelvis fractures, once almost the monopoly of coal miners and construction men, may nowadays happen to any of us who cross the street or ride.

duced by abduction of the leg forcible enough to smash the external tuberosity against the fulcrum of the outer condyle of the femur. One may produce this result, of course, by falling down stairs, but the automobile furnishes the simplest, most fashionable way of doing it. The tire or fender strikes the outer side of the standing leg, with the leg locked in extension, and something *must* give way. It may be, unusually, the outer condyle of the femur. It may be, and not rarely is, the internal lateral ligament, but commonly the tibia gives way first, and the limb is jammed into a knock-knee deformity. It is purely a *crushing* fracture.

Naturally, such a crushing fracture gives comminution, a sort of bone mush, with no real main fragment as a rule. Put up in plaster in the usual way, the leg retains the knock-knee deformity, increasing a bit as the padding shrinks, and the muscles begin to pull; worst of all, the crushed tuberosity, throwing out little callus, furnishes but a poor weight-bearing surface, even at the new angle of weight bearing.†

The *risk* is of a knock-knee with abnormal lateral motion, not very serviceable in walking; and this risk is real.

The *problem* is to return this mashed-out tuberosity to something like normal contour, and to keep it there until it is solidly glued in place, with such added new bone as may be obtained. This means long continued protection.

Attractive as open operation seems, it is not the answer in these cases. I've done a few of these operations, a few years back, one lately, and shall do no more, if I can help it. The results were not bad, nor too good. The trouble is that one finds usually no major fragment to work with, nothing to pin into place. Operation means a certain amount of fresh trauma and reaction, a certain delay in resuming function. My operative cases did well enough as to stability, not so well in the recovery of knee flexion.

We do better, as a rule, by correction of deviation of the limb as a whole, that is to say by correcting the knock-knee to as near a bow-leg as we can manage, and then *jamming* the fragment-mass back where it came from. Thumbs do this pretty well, but I have not scrupled to use a hammer, driving the displaced fragment in and upward (do not forget the *upward* replacement) into position, into such impaction as one may contrive.

Once the fragments are reasonably well in place, one puts on a Thomas splint, with traction enough to hold it in position, and then sets in place two Pearson screw-padplates, one to bear on the internal condyle, the other on the outer edge of the tibia close to the joint: this last pad may press on the fibula moderately, for, after all, the fibular head is usually displaced outward with the tibial mass. Remember the external popliteal nerve, and do not press too low.

†Even far back, when Astley Cooper wrote, he recognized that callus does not form *within* a joint, or that at best, callus formation is much impeded.

This is not a comfortable treatment, but it is tolerable, *and it works*. It should be kept up for about three weeks. After that a plaster "cast" is permissible. (In practice, rather than a plaster, I have preferred a "convalescent caliper" brace with a screw-plate bearing on the inner side of the knee. This has been continued until the x-ray shows completed repair.) Weight-bearing should not be allowed for eight weeks at least. Treated in this way, these fractures do well. Neglected, or kindly treated, they result in knock-knee deformity, sometimes extreme, in instability, and in uselessness.

The neglected cases can be corrected (see case No. 6), but not all will do as well as this one. Case No. 7 is the unfavorable sort of thing we seek to avoid.

It is worth noting that of the fifteen cases, there have been three seen in the past year in which inadequate treatment, or none, has left almost total disability, and an older case (of my own) was equally regrettable, while none of the properly treated cases has shown lateral instability or knock-knee, even if joint motion in flexion was limited to an extent.

There can be, I think, no serious argument as to the importance of treatment, little as to its general form. The routine outline here followed seems satisfactory, and has produced results. Forrester, in his recent book*—appearing since the above was written—has a rather ingenious method of splint-plaster handling. It *should* give earlier joint motion than my routine; as to stability and avoidance of deformity, I have as yet no opinion.

Herewith presented are brief histories of fifteen cases, and four added thereto, in which like trauma produced the *equivalents* of what we are calling "fender-fracture," a name open to criticism, but concisely descriptive, of the trauma at least, chosen because one needs a term to call attention to a class of injuries that seem rather trivial but are fraught with danger of very real trouble.

One may suspect that many of these cases are treated in the routine cast without reduction, and without the especial after-care seemingly essential to decent end-results.

Case No. 1

F. B., building contractor, age 67, weight 240 pounds. Hurt on derrick job, November 11, 1924. Operated on Nov. 14, 1924, with Dr. K. Granger of Weymouth.

Typical fender fracture. Ether: manipulation; forcible remodelling with thumb and mallet, Thomas splint; 2 screw pads. Splint kept on two months.

At three months, still uncomfortable, afraid to bear weight; no deformity.

He recovered slowly; reported as back on his job, despite age and weight, with good function.

Case No. 2

T. M., seen with Dr. Crosbie of Brighton. Age 42. Auto accident. Operation: No main fragment worth pinning in place. Job done with sutures. Plaster fix-

*Imperative Traumatic Surgery, C. R. G. Forrester, Paul B Hoeber, Inc., 1929.

ation. Later, a convalescent splint. Clean healing, comfortable convalescence. No lateral mobility, but marked delay in regaining flexion. When last seen, could flex only to 60 degrees.

CASE No. 3

Mrs. E. C. Age 60, short, weight 200, but vigorous. For two years under treatment for high blood pressure.

FIG. 1. After R. Case 3.

Accident, getting on a street car, May 23, 1927. No reduction done. X-rays taken May 25, 1927, seen with Dr. Edward Stickney at Arlington, June 14, 1927. Convalescent splint prescribed with screw pads. At that time, still some fluid in the joint, some abnormal lateral mobility, no great deformity.

Re-examined July 26, 1927. Motion coming back very well; less lateral mobility. Report of October 16, 1927; splint off, walking very well. Came back to efficient function, for *her* uses.

Died eighteen months later of cardiovascular lesions.

CASE No. 4

Mrs. McV. Age 56. Injured November 11, 1926. Fell from chair hanging curtains; thinks she struck knee on floor. Seen by me with Dr. Pierce McGann, November 15, 1926.

Typical fracture with lateral mobility. Deformity moderate only; reduced next day. Thomas splint with screw pads on either side. Motion after three weeks. Reported later to be an excellent functional result.

CASE No. 5

Mrs. K. Case seen in 1914 with Dr. Frank Davis. Age 58; weight 180 pounds. Had been struck by taxicab. Typical fender fracture, though we didn't call it that then. Seen some days later; no attempt at forcible manipulation, but put up in Thomas caliper convalescent splint. This splint worn for six months.

This poor lady troubled me intermittently until about 1922. There was pain, knock-knee, abnormal lateral mobility, and unfortunately she was not, from age, weight, and cardio-vascular condition, fit for operative relief. No information since 1922.

CASE No. 6

Miss M. F. Age 31. Waitress. Referred by Dr. R. C. Cochrane. Patient fell in a manhole August 5, 1927; sent to the hospital August 6, 1927; discharged September 6, 1927.

Seen by me June 18, 1928. Showed 25° knock-knee, very considerable abnormal mobility, about 15

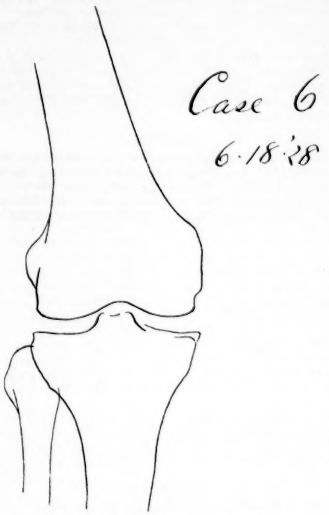


FIG. 2. Case 6: at 10 months.

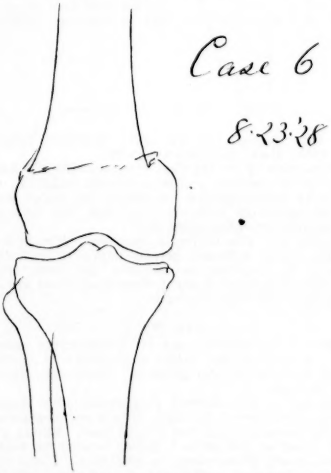


FIG. 3. Case 6. At 10 months after corrective osteotomy.

degrees laterally; a right angle only in flexion, unable to get about without a good deal of pain. Beginning to show definite flat-foot on this side. X-rays shown herewith. Sent to hospital. Operation August 7, 1928: "McEwen's" supracondylar osteotomy.

August 19, 1928, up in chair. Wore convalescent splint for a time, then increasing use.

Seen last March 12, 1929; leg straight, not a trace of abnormal lateral motion; flexion well beyond a right angle. Torn between court action and inertia and the urge to go back on the job, which she seems perfectly fit for.

Case No. 7

Miss J. C. Age 48. Struck by taxicab on February 13, 1928. Examination showed abnormal mobility of knee, laterally. Pillow splint. X-ray showed fracture of external tuberosity—also of fibula just below head. (See x-ray, Feb. 13, 1928, Fig. 4.) High plas-

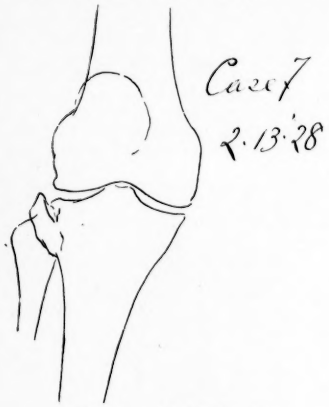


FIG. 4. Case 7: the fresh lesion.

ter cast applied, Feb. 14, 1928. Crutches February 28, 1928; discharged March 1, 1928.

First seen by me the next summer. Showed knock-knee of 30 degrees, grotesque lateral mobility. Ambulatory splint applied, correcting the knock-knee—as a temporary measure. In this, she has gone about very decently, stiff-kneed.

Splint off. She is hardly improved. She is a presenile, rather withered person, happy to be half fit. Should be operated on like the last case, but is content, immovably content, despite all I can say, and is going to go with the splint.

Case No. 8

Mrs. F. Housewife, age 45. Patient not seen by me. X-rays brought (after purloining) by the husband, three days after accident. These seen September 10, 1926.

Showed a very definite knock-knee, 15-18 degrees. Typical fracture of the tibia, with a curious uprising of the fragment, with the fibular head. Fibula probably loose at the ankle. Being treated in plaster. Not a doubt of the ultimate disability. I had to be very circumspect, and husband seems to have been over-ruled, for I never heard from the practitioner.

Case No. 9

Mrs. A. McP. Age 35. Fender fracture in auto accident, December 24, 1928. Typical x-ray.

Referred to me by Dr. Letitia Adams because of

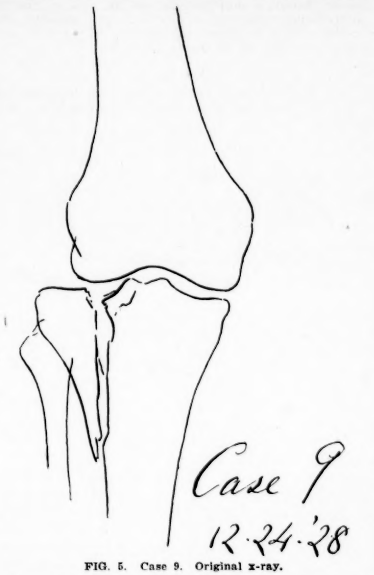


FIG. 5. Case 9. Original x-ray.

unsatisfactory position. Reduced with thumbs and hammer December 29, 1928.

Plaster, presently replaced by convalescent splint, with the two lateral screw pads. Splint worn for two months.

X-rays March 22, 1929, showed admirable position. Clinically, there was no lateral play. Flexion still not over 45-50 degrees. No pain.

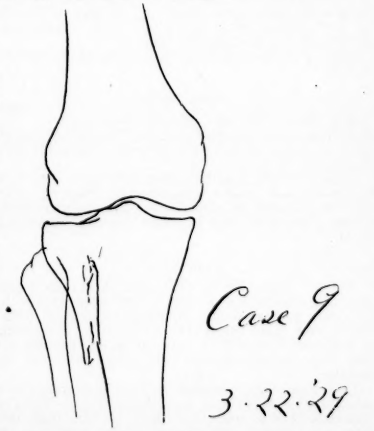


FIG. 6. Same Case 9—after operative (closed) reduction. At 3 months.

Seen July 2, 1929. Flexion now about 80°, and improving. No disability; too content with result to do enough exercise.

CASE No. 10

Miss M. C. Age 44. Typist. Hit by an automobile December 17, 1928. Seen next day by me. Diagnosis

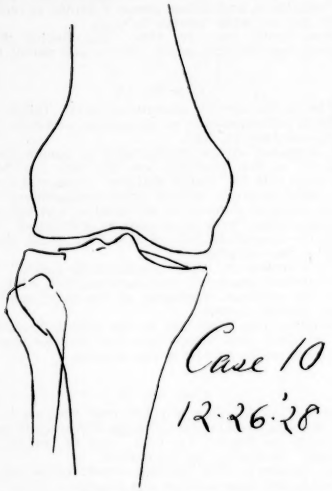


FIG. 7. Case 10. After correction.

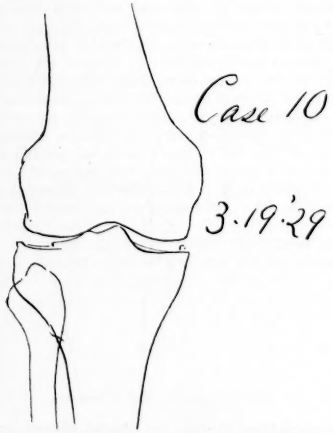


FIG. 8. Case 10. End result; at 3 months.

made, *easily*. X-rays December 18, 1928, showed a fracture of external tuberosity: no great displacement.

December 18, 1928. Ether—reduction; mallet impaction. Immediate application of Thomas splint with the two pressure pads.

December 26, 1928. X-ray examination showed satisfactory position. Splint off at the end of February. X-rays, March 19, 1929, showed a seemingly excellent result. Clinically, on that date, no deformity, no lateral mobility, no pain. Flexion range still limited to about 60 degrees. Excellent function. No pain. In this case a good deal of quadriceps atrophy, with excellent knee motion. Seen July 15, 1929. Quadriceps power improving, instability disappearing. This is muscular instability only,—not a trace of abnormal lateral motion.

CASE No. 11

A. W. Age 69. Injured in automobile accident in which he was run down, not over. Typical fender fracture. X-rays seen December 31, 1928, with Dr. Edward Stickney of Arlington. Typical lesion with a good deal of displacement of fragments, with one rather large fragment. Fibula not fractured. Before I saw the films two attempts had been made at closed reduction without success, in good hands.

Therefore, on January 5, 1929, I operated at the Symmes-Arlington Hospital, twelve days after the accident. The usual difficulty was encountered—much mashed-up bone, not much to pin. In this case there was, however, one large fragment. The operative procedure was to undermine this fragment with small rongeurs, replace it, and drive it home. It was then held with sutures, and a plaster applied after six weeks. Dr. Stickney put him in a supporting brace.

I saw him May 19, 1929. At that time, there was no knock-knee. He could come to 180 degrees extension, 140 degrees flexion, and had a walking leg. Latterly, I have written him but he has not come to see me.

CASE No. 12

F. C. Referred by Dr. Samuel P. Beale of Sandwich, Mass. Age 36, bridge worker. Seen at the Barnstable County Sanitarium, Pocasset, Mass., with Drs. Beale and Wagner.

Accident February 1, 1929. While at work on the

FIG. 9. Case 12. First seen at 3 weeks after. Shows displacement down and out.

bridge over the Cape Cod Canal at Bourne the patient was run down by an automobile. Suffered a Colles' fracture, right, and a typical fender fracture of the left tibia at the knee.

I saw him February 9, 1929. The Colles' fracture needed revision, was revised, and has done perfectly well. The fender fracture of the tibia showed a good deal of comminution, and of displacement. Also, the fibular head was fractured.

Under ether, the leg was manipulated into as near as might be a bow-leg deformity, and with the mallet, I drove the displaced fragment up and inward.

Check x-rays, taken on the spot, showed excellent position. The leg was put up in *plaster*—in "bow-leg" over-correction.

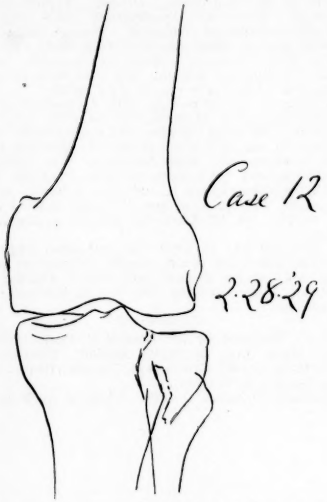


FIG. 10. Case 12. After reduction under ether. In plaster, under strong correction.

At six weeks, a convalescent splint was applied, and the patient set out to walk, protected in walking by the usual screw-pressure-pads, encouraged to mobilize the knee by exercising it, free from the splint, twice a day for mobilization.

Seen at home and examined by me May 26, 1929. There was *no* lateral mobility. There was *no* knock-knee. Extension possible to 180 degrees. Flexion only to 140 degrees as yet. Aspect of joint almost normal. No fluid, no capsular thickening, no pain. Can walk a half-mile, easily. With use (the patient has been cautious), this should be a serviceable, almost perfect joint. Beyond question, it is already a *stable* joint. Seen again July of 1929, flexion to about a right angle, slowly but steadily increasing under exercises. Has a stable and perfectly useful limb.

Case No. 13

M. R., age 64, merchant. Seen with Dr. Thurman January 20, 1929, at home. Run down by automobile, no details. X-rays already taken by Dr. S. A. Robins, showed typical fender fracture of the outer head of the tibia.

Reduction January 21, 1929, at the Beth Israel Hospital. Ether; manipulation into bow-leg stance; impaction with hammer. Plaster, with screw-pad pressure over significant points, from each side. Later, convalescent splint.

Discharged from Hospital February 7, 1929. Examined at office May 13, 1929. Splint long since discarded. Walks almost normally. There is *no* abnormal mobility, laterally. There is *no* deformity. Flexion, as yet, is not over 60 degrees, but there is no bony block, and motion should continue to improve with use for many months to come.

Seen again June 20, 1929. Has flexion fifteen degrees beyond right angle. Stable and useful limb.

Case No. 14

Miss L. M., age 20, amateur aviatrix. Oddly, this aviatrix got smashed in an automobile accident. This happened June 19, 1928.

I happened to be entertaining a pneumococcus, type IV, at the time, but June 20, 1928, I reviewed the films with Dr. Pierce McGann. It showed a typical fender fracture, without great displacement.

Dr. McGann reduced in my fashion, and put on a Thomas splint, with screw-pads as prescribed.

Later—August 28, 1928—I saw fresh films; the reduction was excellent.

On November 10, 1928, I examined the patient. The knee showed absolutely nothing abnormal, clinically, save the slightest smoothing of the joint contours. Joint motion perfect.

Judging from a picture in the Boston Transcript of July 3rd, this young lady has legs as straight and serviceable as before the accident.

Case No. 15

Mrs. N. O'N. This was the case reduced before the clinic at the American College of Surgeons' meeting with us October 11, 1928. Age 68, weight 125 pounds. Mentality, not very good.

On October 9, 1928, she was struck by an automobile; the left knee suffered the damage.

FIG. 11. Case 15. Before handling.

X-ray October 10, 1928, No. 139,700, showed typical fender fracture. Convalescent splint adjusted. After her discharge, in late October, she disappeared. After strenuous search, personally, from Charlestown tenement to Charlestown tenement, I located this lady on June 3, 1929. She had not been to the

hospital "because the boys weren't working." She had continued to wear the splint, having gathered the notion that she was always going to wear it. She was doing the family housework. The joint, without splint, was straight, stable and strong. If she does as I told her, this may in the end become a reasonably mobile joint.

FIG. 12. Case 15. This fragment was pushed and hammered up and in. This is the result at six weeks.

Case No. 16

H. R.: Four years ago a large Scotsman of 60 years hobbled into my office, carefully putting down a foot, and then locking the knee in hyperextension at each step. He was aided by one of the English "racing seat cane" things that one sees not so frequently in this region. The Country Club shows a few. He came to me because I had patched up an arm before and a hand or so earlier. He had been in his capacity as trainer and manager of the Metropolitan Driving Association, a spectator and boss in the exercise of some two-year-old horses. One of these in passing had flung a hoof to the side. As he stood on a hyper-extended knee, this flying hoof had caught him on the lower thigh, and he went down. The lesion was a total tear of ligaments across the inner side of the knee.

Operation, eight days later. The lateral ligament was repaired. The torn crucial was left alone. The ligament tear in this big-boned knee was hardly less than three inches, a clean tear.

The patient is still training two year olds, with a knee as solid as ever; motion in flexion about 15 degrees short of the normal, measured by the other leg.

Case 17

G. P., an Italian 42 years of age. Dislocation of knee in automobile accident. Patient sent to Boston City Hospital. Transferred to me after two months. Reduction of internal dislocation; repair of internal lateral ligament by fascial repair in form of crossed artificial ligament inset beneath cortex above and below joint. He did perfectly well, but when last seen did not have more than 60 degrees flexion.

Cannot be found, despite careful search.

Case No. 18

Negro of 36 caught by automobile, sent to the Boston City Hospital. Rupture of internal lateral ligament—complete. Repaired at 10 days, direct repair without fascia. Kept track of him for eight weeks. Stable joint, but without more than 40 degrees motion. Disappeared, and careful search has failed to reveal him.

The peculiar disadvantage of municipal hospitals seems to be that of false addresses. Apparently twenty per cent. or so of our cases are recorded under names or addresses that cannot be checked up. Seemingly, the psychology is that if one remains unknown, one of the city's weltering mass, one may probably escape payment of board charges. That this involves also escape from the fostering after-care we are trying to provide, does not appeal to this sort of thinker.

Case No. 19

Mr. T., age 32. Automobile accident. Seen with Dr. Cohen December 19, 1928. X-ray showed chip torn off internal tuberosity of tibia, without other lesion. Clinically, some increase in lateral mobility, but not a great deal, perhaps 20 degrees.

Reduced under ether. The chip was pushed into place though not fully. Usual splinting and double screw pads.

Seen May 2, 1929. Has perfectly good joint. Not yet fully mobilized in flexion. Flexion to about 100 degrees.

Seen again June 30, 1929. Flexion to 10 degrees beyond the right angle: no instability, ready for return to work.

NEW ENGLAND SURGICAL SOCIETY

MEDICAL ILLUSTRATIONS OF ANTIQUITY TO THE MEDICAL AUDIO-CINEMA OF THE PRESENT*

BY PHILEMON E. TRUESDALE, M.D., F.A.C.S.†

As far back as written medical records go we find widespread use of anatomic illustration by graphic or plastic representation. In fact, the pictorial and plastic elements precede the written word by many centuries. The earliest of all books on medicine is the "Papyros Ebers", 1550 B.C. But the records in stone and metal go back to 16,000 B.C., to choose a conservative date. Paleolithic drawings of animals with crude attempts at showing bones and viscera were found in the caverns of Ariège, Southern France. Coils of intestine, models of forearm, leg, and head carved in limestone, soapstone, talc, and bone were used by Assyrian, Egyptian, and Babylonian physicians as models for illustration during lecturing.

When lectures came to be written, drawings of these models were used to illustrate the texts. The original statues of the Greek and Roman periods were of terra-cotta, silver, gold, bronze, and marble. Some of the objects found on an island in the Tiber were models of skeletons, thorax, viscera of animals, male and female genitalia. About 350 B.C. appeared a great biological work by Aristotle on "Comparative Anatomy", richly illustrated with drawings. Aristotle, however, never dissected the human body.

About 150 A.D. appeared Galen's great work, "On Anatomical Achievement", in which he described in 16 books the whole anatomical and physiological system. Galen had access to human skeletons, but for dissection he used animals—oxen and apes. Most of the illustrations were made from these animals. From him we get a compendium of all the medical and anatomical knowledge of the time with the different pieces of apparatus and surgical instruments.

After Galen the spread of Christianity with its opposition to science and to the "desecration" of the human body put an end to progress in medicine for more than ten centuries. The ancient diagrams were copied and corrupted, misinterpreted, and used for astrology. Many of these drawings in beautiful colors adorned the leaves of eleventh and twelfth century manuscripts.

The best of Aristotle's and Galen's work with illustrations passed over to the Arabs in the eighth century, and were retranslated during the rise of the Universities in the thirteenth century. Medicine and anatomy had a dignified place in the curriculum, and by 1300 many of the Arabic medical works were translated into Latin. We read of post-mortems at Bologna and the advent of dissection of the human body, although the reason for examining the human body was to obtain information for legal processes, at first. Henri de Mondeville's work on surgery, 1314, and Mondion's "Anothomia" are full illustrations of dissections. The earliest miniature of the fourteenth century at the Bodleian Library shows an operation on a female. The body has been opened from xiphisternum to symphysis pubis, and kidneys, heart, lungs, and stomach are shown, while the operator holds the liver.

With the arrival of naturalism in the fifteenth century we have the beautiful sketches, drawings, paintings, models in stone, bronze, copper, and marble. Michelangelo, Dürer, and Raphael were closely associated with anatomists, and Leonardo da Vinci's art owes much to the anatomical knowledge of his friend, della Torre. All of these artists have left drawings of dissections and anatomical sketches. The greatest was Leonardo da Vinci, who dissected first to improve his drawings and paintings of human beings. His sketches are full of method and technique far ahead of his time. His drawings of the heart, diagrams of shoulder muscles, eyes, urino-genital system, and ventricles of the brain are masterpieces.

Without the models of Leonardo's genius, the work of Vesalius would not have been possible. Vesalius was the first to use living models on whom he used to make the outlines of joints and other parts of the body. Drawings and skeletons were at hand always. He used animals for experiment and dissection. Then criminals were handed over to him and his students to be dissected, and often citizens granted permission for post-mortem examinations on the deceased. His first great work, "On the Fabric of the Human Body (Fabrica)", came out in 1543. Even the initials from the book illustrate the vivisection of a pig, opening of the trachea, and hoisting the body of a dog to a gallows tree for dissection. Some of the most famous of his illustrations are the vascular system, nervous system, dissection of brain, heart, trachea. He compares and contrasts human beings and animals and illustrates likenesses and differences.

The motifs in the "Fabrica" are seen often in works of the Italian Renaissance. Drawings and paintings are the favorite media, since they give the most lifelike representations. Eustachi, physician to the Duke of Urbino, 1574, used

*Address of the President. New England Surgical Society, September 27, 1929.

†For record and address of author see "This Week's Issue," page 1008.

copper engravings entirely to illustrate his lectures, but these were stiff, hard, and inartistic. They were exact and instructive, however, and from these many prints and engravings were made. The Nuremberg school in Germany adopted illustrating by copper engravings, and perhaps the most famous is the series of the developmental history of the human fetus and the child by Coiter, 1573.

The Italians used copper plates and with Guidi we have woodcuts of excellent workmanship representing bones, arteries, veins. From woodcuts and copper plates we can trace the contributions of the period. For instance, in 1572 Varolio gives the name *pons cerebelli* (bridge of Varolius) to the *pons* and the marginal engravings show that he was the first man to examine the brain from its base up.

Often a famous painter like Rubens would draw anatomical sketches in pen and pencil for the copper plates. In 1697 Santorini in a new improved edition of his anatomy used prints of the copper plates done in a light crayon effect to bring out the difference in the tissues. Anatomic representations now appear in colored woodcuts.

Illustrations in mezzotinting, by three different impressions—blue, yellow and red, were made as early as 1704. The French became very proficient in the making of colored prints, and d'Agoty of Marseilles added a fourth black plate to bring out the colors and create a more striking effect.

John Brisbane, an Englishman, wrote in 1735 that good illustrations may often serve the purpose as well as or better than personal dissection, and pleaded for more good drawings for the general information of the layman. He made a short collection of models and wrote a brief, introduction to anatomy replete with illustrations.

Lelli, director of the medical academy at Bologna, 1766, used wax models and copper plates to illustrate his lectures. Adam, 1773, published fifteen myologic and osteologic copper plates very beautifully done. For nine of them he used red crayon.

The best plates, however, are those of von Soemmerring, who aimed for artistic reproduction with exact perception of details in every part of the human body. He trained stucco-workers, modelers, and draftsmen to work for him, and had them experiment with sepia and pigments, and told them always to have in mind the motto, "Attain to nature's perfection." Much of the work contains criticism of former illustrations on the same subjects. By this process anatomic illustration is improved. Soemmerring's most perfect work was a series of beautiful colored plates on the human eye.

Wax models seem to be the only other medium of illustration besides the colored copper plates during this period, and from 1800 on, the plates grew more beautiful, most of them being made from nature. Highly finished lithographic impressions adorn the work of Simpson, Flaxman and other English surgeons. Many were finished in crayon or colors on India paper. Some were lithographed directly without being first engraved on copper. By 1800 many rare editions appeared in England, Germany, France and Italy. Many authors had copper plates made from their own sketches in crayon, ink or water color. Statues in bronze and marble were drawn and the drawings used for the making of lithographic plates. Indeed, material for the plates were obtained from any plastic or graphic source. Salomen based a series of copper plates on Houdon's anatomical plaster statuette, and figures in wood and wax were used as models repeatedly. Some of the plates were in black and white, others in colors. A conception of the multiplicity of these illustrations, copper vignettes, etchings and engravings can be obtained by perusing Choulant's illustrations, the most admirable of which were those of Soemmerring.

In the presentations of his remarkable illustrations of the vital organs of the living body Soemmerring approached the natural interpretation in a more masterly manner than could be found in the works of any of his predecessors. He was far in advance of his time in portraying a vivid texture in his illustrations of anatomical structures. Apparently his pen and pencil impressions, although of exceptional excellence, lacked two important features which modern science has contributed in the form of motion and voice reproduction.

In 1845 the zoetrope was developed. It consisted of a cylinder revolving on a vertical axis and having a number of vertical slits cut near the top. On the inside surface of the cylinder was placed a strip of paper having on it a series of pictures representing an object in successive stages of motion, as a horse running. An observer looking through the slits at the moving picture inside the cylinder saw the object in motion repeating the cycle of movement with every revolution of the apparatus.

In 1877 Eadweard Muybridge in America made a series of photographs of horses in motion, employing dry plates, because at this time sensitized films had not been invented. The horses were photographed at various points of their progress around the race track by placing along the track a battery of cameras in proximity, the shutters of which were operated by strings broken by a horse as it passed along. At the time these pictures were made they marked a decided advance in the art of photographing objects in motion.

The kinetoscope brought out by Edison in 1893, and independently developed in a modified form by Lumier, Paul and others shortly afterwards, comprised a machine operated by hand or electric motor for moving a transparent film in front of an aperture through which an observer might view a consecutive series of pictures by the

aid of a lens, suitable illumination of the film being provided. A modification of the apparatus allowed the image of the film enlarged to be thrown by a suitable lantern so as to form magnified images of the photographs on the screen. This, in essence, is the modern moving picture machine. When projected at the rate of 16 to 20 pictures per second, one visual impression does not entirely die out before another succeeds it, and the illusion of actual motion results.

While many inventors have made machines and devices for connecting moving picture projectors with talking machines, many of which have been extensively exhibited, it is difficult to make the combination a satisfactory one. Edison's kinetophone first put before the public in 1912 proved defective in the quality of voice reproduction and sometimes lacked in synchronism. Gaumont developed a type of apparatus for electric control of one machine by the other. The result was uniform operation of the two by means of a current obtained from the armature of a motor generator attached to the operating shaft of the phonograph. Such mechanical connections for voice reproductions, however, have been practically abandoned.

The talking motion picture which is about to be presented, in all probability foreshadows the introduction of a new personality in education, the audio-cinema teacher in medicine. Making due allowance for the infirmities of the author of this picture as an apprentice, one cannot expect it to be considered more than primitive in the light of developments which already suggest themselves. In the hands of teachers of experience and gifted expression rapid progress may be expected. As it is today, the structure rests upon a tripod, one leg of which is represented by the investigations, observations, experience and association of ideas of the author; the second leg affords support by animated reproduction of pictorial and plastic illustration, while the third maintains a footing by means of the audio-cinema evolved through a synthesis of visual motion and vocal description.

I realize fully that in order to give it precedence to or even rank with present methods of teaching, there is need of much patient study, co-öperation and endurance. For example, an anatomical still picture without color is not as intelligible as one with color differentiation. When the subject is in motion, it is *less* clear. Blood is black; muscles, vessels and nerves are seldom distinct. The slightest shadow produced by the hand or instrument dims the operative field on the film. Therefore, problems of posture and light require strict attention to detail. We have pictured four thigh amputations, and although the last is better than the first, we have not produced one film worthy of presentation. It is well to remember that in presenting a medical subject in motion pictures, realism may defeat its own purpose by being too realistic.

Moreover, it may be necessary to modify the atmosphere of the studio. The doctor is essentially a teacher, not an actor. While there are certain qualities peculiar to and essential for both, the doctor is primarily a school man and the actor a dramatic artist. The doctor's position is that of a messenger from a scientific body. He seeks to present clinical and experimental evidence in support of his observations. His attitude, personality and power of expression are essential for convincing argument, but a make-up which gives the doctor a school boy complexion and the mien of the happy warrior makes him a product of decorative art, not a disciple of Galen.

A much desired effect of the talking motion picture in presenting a medical subject will be the economy of words, since every syllable represents a monetary tax upon the speaker, for the first time in history there will be this very tangible controlling factor in favor of brevity without loss of substance. With every unnecessary word deleted from the text, the recital of the teacher by phonetic and artistic description converges upon the point at issue in such a manner as to engrave a lasting impression upon the memory of the student.

Furthermore, you are aware of the plight of the general practitioner in his struggle to keep informed upon the recent advances in medical knowledge and their practical application in his daily program. By the monetary cost of travel in pursuit of post-graduate study, and that intangible, but nevertheless very appreciable loss in clientèle entailed by absence from his community, the doctor's continuation schooling is reduced to medical books and journals. In the light of such developments of modern science as we shall see in this picture, it is likely that certain relationships between teacher and students of medicine will be modified. Lectures and demonstrations recorded on films will be made available to doctors wherever located, in city, town, or backwoods. Instead of the student appearing before the teacher in his laboratory or in the amphitheater, the teacher and his amphitheater or the laboratory appear on the film before the doctor at a society meeting, a hospital staff conference or, it may be, in the doctor's own library. Indeed, a look into the future cannot fail to excite inspiration over the inherent possibilities of this new relationship of science, art and electricity in the field of medical education.

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The subject of Hernia of the Diaphragm with the mechanism of transposition of the abdominal and thoracic organs was demonstrated by means of a talking motion picture.

ADDRESS TO NEW ENGLAND SURGICAL SOCIETY*

BY MR. F. H. ARLINGHAUS†

MY associates and I are truly grateful for this opportunity of speaking to you about our opinions concerning the possibilities that talking motion pictures offer the medical profession.

It may be well to say something about the early struggles involved in the development of talking motion pictures. They were conceived and developed by the Bell Telephone Laboratories, an organization given entirely to the research in the fields of sound amplification and sound transmission. Many years of patient effort against many unsuccessful attempts finally produced apparatus which gave them the quality for which they had been striving—quality of sound recording and quality of sound reproduction. The matter of synchronizing sound with motion was merely a mechanical detail and was not long in being secured after the most vital characteristic — quality — had been obtained. The equipment, as designed by the Bell Telephone Laboratories, was turned over to the Western Electric Company for manufacture.

The introduction of sound motion pictures to the motion picture industry created a radical change in such a short period of time that we almost feel justified in saying that its effect was revolutionary. We do not think that it is necessary to spend much time talking about the success of talking pictures in the theatrical field, as it is only necessary to look about us to recognize the inroads that this new means of communication has made on an industry which had been considered as very stabilized.

The modern scheme of education as followed in our schools and universities, plans to associate the theoretical with the practical so closely, that the result will be a sound, thorough, knowledge of the subject at hand. This association of the practical with the theoretical usually requires the simultaneous functioning of two senses. The most popular combination of senses utilized for the gaining of knowledge, is that of sight and hearing. The attention of this combination of senses is demanded by talking motion pictures to absorb the information which they contain. We therefore feel that talking motion pictures are to become a vital factor in the plans of education in the future. At any rate, we are sure that they represent a very potential medium for communicating information from isolated sources to groups of individuals.

Realizing that a thorough knowledge is a short cut to success, we have therefore appealed to physicians to secure their opinion as to whether talking motion pictures could assist the medical profession, and if so, in just what way. To concisely express the consensus of these opinions, we might say that in general they believe that talking motion pictures can be utilized by the medical profession to greatly assist the average practicing physician in keeping abreast with the progress made in the field of medicine. They point out that the average physician is eager to continue his studies, but is at a loss as to just how to go about it, for the reason that little or no time can be taken from his practice without material loss to himself. It therefore necessarily comes down to the amount of spare time he can manufacture for the purposes of reading or attending lectures in nearby centers. This, at best, is a slow process for the acquiring of new and additional knowledge. They are outspoken in their opinion that in selected medical subjects it would be to their advantage to be able to witness new methods of practice in the form of motion pictures synchronized with instructive and appropriate lectures.

Our initial efforts have been greatly assisted by the foresight and thorough co-öperation of Doctor Truesdale, and our gratitude to him is such that it becomes difficult for words to adequately express. He has produced for us a talking motion picture whose contents are of interest solely to the medical profession, and has consented to the first installation of equipment for reproducing sound motion pictures in hospitals. This equipment which is here today, is to be installed in the Truesdale Hospital at Fall River. Dr. Truesdale has done all of these things in whole-hearted fashion and has not asked for consideration in any form for his pioneering efforts to establish talking motion pictures in the medical profession.

We are frankly hopeful that the spirited initiative of Doctor Truesdale will be an incentive to hospitals, doctors, and associations to immediately contribute to this program outlined for the benefit of every physician in this country.

Now we wish to publicly acknowledge our indebtedness to Doctor Truesdale for his valuable assistance, for his invitation to attend this meeting, and for the privilege of expressing our opinions on this subject. We thank you Doctor, for all these, and you, gentlemen, for your kind interest and attention.

*Delivered September 27, 1929.

†For record and address of author see "This Week's Issue," page 1008.

Case Records
of the
Massachusetts General Hospital

ANTE-MORTEM AND POST-MORTEM RECORDS AS USED IN WEEKLY CLINICO-PATHOLOGICAL EXERCISES

EDITED BY R. C. CABOT, M.D.
F. M. PAINTER, A.B., ASSISTANT EDITOR

CASE 15461

EIGHT WEEKS' FEVER IN A FEEBLE-MINDED GIRL

MEDICAL DEPARTMENT

A thirteen-year-old colored girl was sent into the hospital March 25 from one of the schools for the feeble-minded because of fever of a week's duration. Her intelligence was of such low grade that the only information obtainable was from a brief note from the institution and the story of the mother, whose replies were evasive and apparently unreliable.

March 18 the girl's temperature was 100°. She had no tenderness and did not complain of pain. March 22 the temperature rose to 104°, still apparently without pain or tenderness. A low enema gave good results and she seemed better. "Today (March 25) the distention has increased and she has tenderness. Temperature ranged from 100.4° to 103.4°."

She had been mentally defective since birth. She was incontinent of urine from early childhood until a year or two before admission, so that she could not go to school. Two years before admission she was brought to the Out-Patient Department because of a rash about the vulva. Examination showed adenitis of the cervical, epitrochlear and inguinal glands. She had a history of delinquency,—theft and attempted incendiarism. A brother was also in an institution for delinquents, and a sister was very backward. The father had "spinal rheumatism" and "bad kidneys." The family were supported largely by the overseers of the poor.

The patient had measles at two years, chicken pox and ataxia at three. At that time she was sent to Long Island, where she stayed for a year. Her legs were very edematous and had to be "pumped out." At five she had scarlet fever. She very often had dysuria; sometimes screamed with the pain. She had had hematuria once or twice. From time to time she had "lumps around the neck." The summer before admission one of these was removed from the back of her head. She had not yet menstruated.

Clinical examination showed a thin, "pale" colored girl, drowsy but conscious. Skin clear. Diaphragm possibly pushed up. In the back slight dullness to the angles of the scapulae. Purulent exudate in the nasopharynx. A few enlarged posterior cervical and left axillary glands. Apex impulse of the heart seen and felt in the fourth space. Left border of dullness 7.5 centimeters from midsternum, one-half centimeter outside the midclavicular line. Right border doubtful. Supracardiac dullness 4. Sounds and action normal. A questionable systolic murmur at the apex. An early diastolic. Blood pressure 105/50. Abdomen distended and tympanitic. Questionable slight shifting dullness in the flanks. Extremities, pupils, reflexes and fundi normal.

Amount of urine sixty-two to eighty-eight ounces March 28 to 30; otherwise amount normal when recorded. Specific gravity 1.010 to 1.032. The slightest possible trace to a trace of albumin at six of twelve examinations. A few to occasional leukocytes at six of nine sediment examinations. No red cells or casts. Blood: 8,400 to 22,300 to 2,200 leukocytes, 85 to 92 per cent polymorphonuclears, hemoglobin 75 to 80 per cent, reds 4,230,000. Smear at entrance normal. Hinton negative. Urine culture negative for typhoid bacillus. Two Widals negative for typhoid and paratyphoid A and B. Vomitus: guaiac negative. Stools: guaiac positive at one of nine examinations.

Temperature 97.4° to 105.3°. Pulse 86 to 150. Respirations 20 to 30, after April 16 normal. X-ray examination showed the diaphragm very high and sharply domed on the right. It moved with respiration. The hilus shadows were moderately increased, and there was some soft mottling around the root of the right lung. The interlobar septum was visible on this side. The changes in the lungs were slight and perhaps of no pathologic importance. The high position of the diaphragm suggested fluid in the abdomen.

April 2 a blood culture showed in both flasks a Gram-negative bacillus the cultural reactions of which were those of typhoid bacillus, but which did not agglutinate with the corresponding antiserum. The abdominal distention increased. April 6 the temperature dropped without discoverable cause from 104° to 99°, with slight elevation of the pulse and respirations. April 14 there was dullness without other signs at the left base. April 16 this was more marked, the breath sounds were diminished, and no tactile fremitus was obtained on either side. Another blood culture showed no growth. The next day a chest tap in the eighth left interspace gave 30 centimeters of clear fluid, specific gravity 1.020. Ziehl-Neelson stain and guinea pig inoculation showed no tuberculosis. A bacteriologic consultant found quite marked tenderness over the left kidney, probably not related to the chest tap wound. He thought the organism shown in the culture was not typhoid, but merely an incidental organism in the blood stream.

The temperature remained high. April 20 the right base showed a few crepitant râles. The left base showed dullness with diminished to absent breath sounds and diminished spoken voice. The patient was tender all over,—on the tip

of the nose, the tips of the toes, over the scapulae, etc. The fundi were negative as far as could be determined. It was now thought that the fluid obtained at the tap might have come from the abdomen, not the chest. A heart consultant found only a short early diastolic murmur along the left upper sternum.

May 2 the lungs were clear. May 3 the abdomen showed soft distention, with much tenderness in both flanks. There were coarse crepitant râles at the right base. May 11 the patient was very weak and drowsy. The blood pressure was 80/50. There was slight tenderness in the right lower quadrant and just above the pubis. May 13 the spleen was three centimeters below the costal margin. The patient retained nothing by mouth and was very emaciated. A portable X-ray showed no gross changes in the lungs. A surgical consultant thought operation not indicated. May 16 the patient died.

Discussion

BY RICHARD C. CABOT, M.D.

NOTES ON THE HISTORY

I should think we have about all the obstacles that could be piled up against getting a satisfactory history.

Distention of the abdomen I suppose which was previously present is recorded "increased." The only point of stopping over these lapses in histories is that you will find when you are writing histories that you yourself leave out something that you meant to put in. We are constantly finding even in our histories, which are carefully written, such expressions as "the patient had pain in the right side." The man who writes it knows that he means the right side of the chest, not the head, abdomen or buttocks. When we are writing a history we should put ourselves in the place of the person reading the history; otherwise we leave out essentials.

You can guess as well as I as to what "spinal rheumatism" and "bad kidneys" might mean. A possible guess is tabes, which would give him urinary frequency, the symptom on which the lay diagnosis of "bad kidneys" is usually based. "Spinal rheumatism" might be the lightning pains of tabes. That is all very speculative.

"Ataxia" probably means that being feeble-minded, at three years she did not walk as other children do.

Certainly the edema of the legs is an important piece of information if true. There is nothing in feeble-mindedness so far as I know to produce edema of the legs. We think of heart disease, kidney disease or both. About the "pumping out" of her legs, you know there is a way of getting fluid out of the legs by putting hollow needles into them. I do not know that I have ever heard of using them on so young a child, but I do not know why they should not be used. This may mean the use of diuretics or something of that kind. It depends upon the degree of imagination of the person who wrote this history.

Perhaps later we shall get something that will give us hints about that hematuria. We first think, "Did it ever occur?" People often speak of bloody urine when the fact is they have a high colored urine. Our information comes from such unsatisfactory sources that I doubt whether she did have hematuria. A stone in the bladder is a common thing in a child of this age, but is likely to give much more steady bladder symptoms than have been suggested in this history. It is quite possible that we shall never know about that hematuria, and quite possibly it never occurred.

NOTES ON THE PHYSICAL EXAMINATION

Presumably the "lumps around the neck" are enlarged glands from infections in the mouth.

Pallor is very hard to tell about in a negro. We have to tell from the mucous membranes, and even those are brownish. Unless we are practising among negroes all the time it is hard to be sure.

I suppose the dullness to the angles of the scapulae made them think that the diaphragm was pushed up.

On the whole I do not see why we should suppose any cardiac abnormality from the heart measurements.

An early diastolic murmur is important if true. This is a rather large pulse pressure, which might go with early diastolic at the apex.

The important thing in the examination is that she has no edema of the legs, which is reported to have been extreme before. She may have edema in the lung cavities, in the pleurae or the peritoneum. We are not sure in either case.

In any case the thing I want most to know about a urine in relation to kidney trouble is the amount of variation in specific gravity. We have an excellent variation here. We cannot ask for any more. So far the evidence is in favor of the kidneys being sound.

We are not told whether those urine records were from catheter specimens. We have no reason therefore to put any special dependence on the record of sediments.

We have very little so far except an early diastolic murmur once recorded and some fluid in the chest and abdomen. We are not sure of those last statements.

The temperature rules out typhoid, because in typhoid, except at the very end, we never get temperature as low as that.

The X-ray shows no evidence of fluid in the chest. The costophrenic angles are clear.

You can take your choice as to whether the culture showed typhoid or not. I am going to choose non-typhoid.

The chest examination April 16 sounds more like pushed up diaphragm or fluid than it does like solid or pneumonia.

Thirty cubic centimeters of fluid is not worth

tapping for. But 1.020 is a strikingly high gravity, not like that of an ordinary dropsical fluid. If that record is correct it makes us suspect tuberculosis, tuberculous pleurisy being the commonest cause of fluid with a gravity as high as that.

I agree with the bacteriologist. I do not think they would have got 30 cubic centimeters out of the kidney anyway. The eighth space is pretty low for a tap, you know. One is pretty uncomfortable about one's whereabouts when one is as low as that.

Tender toes are associated with what disease? It is a typhoid sign discovered by Sir William Osler, who as he walked along his ward used to put his hand on the feet of each patient in bed. He could not shake hands, so he would shake feet. When patients winced he noticed they were more apt to have typhoid. But here there is tenderness in so many places that it probably is not significant.

It is quite possible that the fluid was from the abdomen, but not from the kidney I think.

That is the same murmur, as I understand it, that we heard before.

The temperature chart is distinctly worth looking at. We cannot blame anyone for thinking of typhoid when we see elevation as steady as that. That is running along from 103° to 104° for over two weeks. It went down for a little while, then up again.

DIFFERENTIAL DIAGNOSIS

We have a thirteen-year-old girl who after a fairly short history, a week's history so far as we know, is brought here and lives for seven weeks. It is fairly safe to say she had an infectious disease. The question is, what infectious disease?

In 1907 I studied 784 cases in this hospital that had shown two weeks' fever without touching normal to any extent.* Out of these 90 per cent fell into three groups according to causes. I have found that classification a very useful one ever since. Long continued fever that does not touch normal to any extent generally means typhoid, tuberculosis or sepsis, using sepsis in the wide sense that includes malignant endocarditis or any fever that goes with an abscess or a blood infection not localized. Therefore seeing that chart my first impression is that the case ought to fall within one of those three disease groups. We will first go over those before we think of others.

1. Typhoid certainly suggests itself when we look at the chart. A typhoid chart shows a relatively low pulse. So does this. This pulse runs along from 90 to 100 most of the time when the temperature is 103° to 104°. She comes in at the time of year when typhoid fever is not very common. In Massachusetts typhoid fever has been pretty nearly ruled out by good water supplies. The spleen was enlarged at the end, but apparently not so at the beginning. There is no record of rose spots. In a negro they are hard to see. Therefore we should not count that. There were two negative Widals and no good bacteriologic evidence in favor of typhoid. She died too soon for uncomplicated typhoid. Typhoid patients die either of a very long fever which wears the patient out, or of complications. Complications are usually hemorrhage in the bowel or perforation of the bowel.

A STUDENT: She was in the hospital fifty-two days.

DR. CABOT: I am glad you pointed that out. I did not realize it was so long. I take back what I said about her dying too soon for typhoid. I do not think that is proper reasoning.

Typhoid should not show fluid in the abdomen or anywhere unless there are complications. Apparently she did have fluid in the abdomen. I think it was a little of that they got when they tried to tap her chest.

2. Now tuberculosis. She is of a race in which and at an age when peritoneal tuberculosis is not rare. If she had fluid in her abdomen and if it had a high gravity, as seems to be the case, that supports the idea of tuberculosis. The fact that they did not find the tubercle bacillus in the fluid is not important. They did not have much to deal with. Abdominal tuberculosis, peritoneal tuberculosis generally shows some tenderness. She had some tenderness, though it does not seem to be as generally distributed as we expect it to be in peritoneal tuberculosis. I can go no further in relation to the diagnosis of tuberculosis. I do not see how it can be ruled out. If she has it I think it is chiefly abdominal, which would be peritoneal, and probably intestinal as well. The absence of diarrhea does not rule that out.

A STUDENT: How about renal tuberculosis?

DR. CABOT: Renal tuberculosis is one of the things we ought to consider. She is said to have had hematuria. But pyuria is commoner in renal tuberculosis, and she did not have anything we could call pyuria here; the urine would have shown leukocytes; and the great majority of diagnosed renal cases have painful bladder symptoms. She had some tenderness over the kidneys. But the kidneys were never felt. The diagnosis of renal tuberculosis has to rest on feeling a kidney and on the urine. We have not much evidence of it. When I say abdominal tuberculosis I mean of course mesenteric glands as well as peritoneal and very likely intestinal. There may be some tuberculosis in the chest. I do not think the physical signs are enough to enable me to decide about that. But I do not believe the main thing is pulmonary tuberculosis, if it is tuberculosis.

Did she have a miliary tuberculous process? She was drowsy, but I do not now recall any other cerebral symptoms. A miliary process generally gives more definite cerebral symptoms, has to find a focus of sepsis unless one finds evi-

*R. C. Cabot. The Long Continued Fevers of New England. Boston Med. & Surg. Journal, Aug. 29, 1907.

dence of it in the blood stream. I cannot find a focus in my memory of this history. It is hard to say anything about her blood. We do not know the relation of the differential count to the total counts. She certainly had one high total count and one high differential. That does not rule out tuberculosis. It is perfectly consistent also with sepsis.

Are there any other infections we ought to consider? Of course grippe, influenza, is to be considered in almost every case that shows fever. But I never heard of a case running so long a fever. We have no evidence of exanthemata or of any malignant disease such as can cause fever, or of syphilis. There is little in the history to suggest syphilis, and nothing in the examination at the present time.

On the whole it seems to me more like tuberculosis, and as I said peritoneal tuberculosis, than anything else I can name.

A STUDENT: Would you consider endocarditis following scarlet fever?

DR. CABOT: Endocarditis of course can follow scarlet fever. The scarlet fever was eight years ago, and I should be surprised if endocarditis after scarlet fever in a child could last as long as that. We have this diastolic murmur which I am glad you brought me back to consider. I do not know what to make of that. It is conceivable that it may be the basis of the whole trouble. She may have vegetations on her aortic valve and fever from that. But we have no evidence of embolism and we have no blood culture. We have not the type of fever that usually goes with septic endocarditis. I cannot rule it out, but ordinarily the evidence is clearer than it is stated to be here. I am voting on the whole against it for the reasons I have given.

A STUDENT: Could it be undulant fever?

DR CABOT: What used to be called Malta fever? We cannot go very far without more evidence. It does not seem to me likely. That is a rare disease in this part of the world.

DR. TRACY B. MALLORY: Two cases have been called to our attention recently by members of the staff of this hospital.

DR. CABOT: I had not heard of them. It is a very rare disease here, and in the absence of bacteriologic evidence I do not see how we can say anything about it.

A STUDENT: Could it be colon bacillus infection?

DR. CABOT: I do not believe so. That is also rare. We did not get it in the blood, and there are some more definite changes in cranial nerves than are shown here.

3. Sepsis is the third natural possibility. One an easily cultured organism. I think we can rule it out.

A STUDENT: Would you consider abdominal malignancy?

DR. CABOT: Lymphoma is perfectly possible at her age. But it is queer if we had not felt anything. After this number of days it seems as if we should have felt some abdominal tumors. I do not think it is probable.

A STUDENT: How would you account for the generalized adenitis?

DR. CABOT: It was not found during the present year, was it? I think I will pass that.

A STUDENT: How do you account for the enlarged spleen?

DR. CABOT: That could perfectly well make part of the picture of tuberculous infection.

A STUDENT: Would syphilis show as high a fever as this?

DR. CABOT: I think not. I have never seen it.

CLINICAL DIAGNOSIS (FROM HOSPITAL RECORD)

Tuberculous peritonitis.

DR. RICHARD C. CABOT'S DIAGNOSIS

Tuberculous peritonitis.

ANATOMIC DIAGNOSES

Tuberculous peritonitis.
Tuberculous pleuritis.
Chronic tuberculosis of bronchial lymph nodes.

DR. MALLORY: The case was one of tuberculous peritonitis. The peritoneal surfaces were studded with tubercles and there were between 300 and 400 cubic centimeters of quite turbid yellow fluid. The spleen was considerably enlarged, weighing 150 grams. That is very large for a girl of this size. It did not show any tubercles grossly, however. The right pleural cavity did contain some fluid, about 150 cubic centimeters, but no evident tuberculosis in the pleura on that side. The left pleura, which did not show any fluid, showed tubercles. There were lesions found on the outside of the intestines, presumably simply part of the tuberculous peritonitis, but no lesions of the mucosa of the intestines. The primary focus of the tuberculosis in this case was pulmonary. The bronchial lymph nodes were considerably enlarged and caseous.

DR. CABOT: The intestines were negative, too?

DR. MALLORY: Yes.

A STUDENT: Were the meninges and spinal cord involved?

DR. MALLORY: They were not examined.

DR. CABOT: There was nothing in the heart?

DR. MALLORY: No.

A STUDENT: Was there anything suggesting syphilis in the spleen?

DR. MALLORY: No.

Everything else was entirely negative.

CASE 15462

SECONDARY ANEMIA WITH MENORRHAGIA; COMPARATIVE RESPONSE TO LIVER AND IRON THERAPY

MEDICAL DEPARTMENT

Present Illness: An Irish-American district nurse aged 40 reported for a health examination on August 22, 1928. She came purely for routine examination and was not seeking advice.

She complained of no symptoms voluntarily, but upon questioning admitted that she had been feeling rather tired for about six months and that catamenia had been abnormal. Periods were rather profuse for the last three months and had occurred every two weeks, lasting usually more than a week. She was also about five pounds below her usual weight and her appetite was not good. She had been a little dyspneic on exertion for about two months. She had never had sore tongue, diarrhea or tingling of her hands or feet. Except for the menorrhagia, there had been no visible loss of blood.

Past History: Patient's health had always been good. She had had no operations or serious illnesses. She had whooping cough in childhood, tonsillitis once about four years ago, and was apt to have one or two colds each year.

Physical Examination: Weight 114½ pounds; general development and nutrition good, but color of skin and mucosae was very pale. Skin clear, except for pallor. Hair light brown, with a few patches of gray. Eyes normal. Throat, negative. Teeth all removed; wearing upper and lower plate. Tongue distinctly smooth around the edges. Thyroid normal. Glands normal. Heart normal in size; sounds regular; rate 118, good quality, no murmurs. Blood pressure 144/80. Lungs clear. Abdomen level, soft, tympanitic; no tenderness or spasm. Liver edge could be distinctly felt to descend about two inches below costal margin in nipple line. The tip of the spleen was easily palpable on deep inspiration. Reflexes and extremities normal except for a few varicose veins.

Laboratory Examinations: Urine normal in color, specific gravity 1.008; no albumin; no sugar; sediment negative. *Blood:* hemoglobin 40 per cent, Sahli; reds 3,070,000; leukocytes 4,100. The stained specimen showed distinctly increased variation in size and shape with occasional very large or very small cells. There was a slight general achromia; the platelets appeared normal; polynuclears 63 per cent; lymphocytes 24 per cent; large mononuclears 6 per cent; basophiles 1 per cent; atypical cells 6 per cent, probably all large mononuclears; icteric index 6; Wassermann negative. Stools: two specimens were negative for blood and parasites. Gastric analysis August 25 as follows:

	Free hydrochloric acid	Total acid
Fasting contents	0	8
Test meal at 15 minutes	6	15
" " " 30 "	25	43

Discussion

BY JAMES H. TOWNSEND, M.D.

The exact type of anemia present in this case is not entirely clear. That it belongs in the "secondary anemia" group seems fairly certain, however. The low color index, the achromia and the general character of the microscopic picture place it in this class. The possibility of primary pernicious anemia is suggested by the smooth tongue and the enlarged liver and spleen, but the patient had had none of the characteristic sore tongue or spinal cord symptoms, and the finding of the free hydrochloric acid in the gastric contents definitely rules out this diagnosis.

Whether the anemia is secondary to the menorrhagia or the menorrhagia secondary to the anemia is not clear. That the two are intimately connected is strongly suggested by the subsequent course of events. When her blood is up to normal her periods are of normal duration and amount, and the first indication that she has anemia is menorrhagia. The two seem to form a vicious circle.

A pelvic examination was made to rule out organic cause for bleeding, and nothing abnormal was found. Curettage was planned, but later improvement made this unnecessary. Other causes for anemia which were considered were:

1—Disease in the gastro-intestinal tract.

2—Banti's disease.

3—Atypical leukemia.

A gastro-intestinal cause was ruled out by the lack of symptoms and the negative stools. It was not considered necessary to take X-ray pictures. The spleen was hardly large enough to make Banti's disease at all likely, and the subsequent disappearance of the spleen practically rules out this diagnosis. Atypical leukemia is suggested by the presence of the atypical cells in the blood smear. This possibility must be kept in mind until a number of years has gone by, but to date there has been nothing further to suggest it.

Further History

Treatment and subsequent course.

A diagnosis of secondary anemia due to the blood loss from menorrhagia was made, and the patient was started on treatment August 25, 1928. Her dietary habits were gone into carefully and it was found that she had been taking an average normal amount of iron-containing foods. She was in the habit of taking meat once every day, and of eating always one and usually two vegetables, including green leaves, two or three times a week. No striking defects were found in her diet. She was advised to take raw liver, one-half pound daily, strained and mixed with milk, and was given iron in the form of ferrous lactate, five grains twice daily. Her response during the next four weeks is shown in Table 1.

March 15, 1929: Patient had neglected to report for over four months, but stated that she had felt very well and the only illness was a slight cold. She had given up the raw liver and iron soon after the last visit, but continued to eat cooked liver about once a week. Her appetite and strength continued good. Her periods

had been normal in December and January, but the last one had come ten days early. On examination she did not look quite so well and was noticeably pale. The spleen was just palpable, but the liver could not be felt; no glands; no jaundice. Weight 125; hemoglobin 60 per cent; reds 4,490,000; leukocytes 7,000; smear, polynuclears 44 per cent; lymphocytes 43 per cent; large mononuclears 5 per cent; eosinophiles 5 per cent; platelets looked normal. She was advised to go back to the ferrous lactate, five grains twice a day, but to omit the liver. This time her response is shown in Table 2.

TABLE 1

Date	Weight	Treatment	Hemoglobin	Red cells	Leukocytes	Catamenia
August 28	114½	None	40%	3,070,000	4,100	Profuse
Sept. 7	114	½ lb. liver and 10 grains ferrous lactate	45%	4,170,000		Nor.
Sept. 21	116½	"	70%	4,680,000	6,500	Nor.
Oct. 6	119	"	80%	5,140,000		Nor.
Nov. 1	121½	"	85%	5,200,000	11,000	

August 25, 1929: Patient became tired of iron soon after last visit and gave it up. Except for this her habits were unchanged. She continued to get meat daily and to have a liberal amount of vegetables. For the last week or two she had felt almost as tired as when she was first seen. Periods had come every two weeks lately and she had been flowing steadily for fifteen days. Weight 122½. Hemoglobin 50 per cent; reds 3,220,000; leukocytes 8,000; reticulated cells 2.6 per cent. Smear, polynuclears 61 per cent; lymphocytes 25 per cent; large mononuclears 8 per cent; eosinophiles 2 per cent; atypical 4 per cent; red cells showed slight achromia, marked anisocytosis and poikilocytosis, a few stippled cells were seen. The patient was told to go back to iron and ammonium citrate, but take no liver.

Sept. 3, 1929: Not much change, still flowing. Hemoglobin 50 per cent; reticulated cells 1.8 per cent. On account of the slight response of the reticulated cells it was decided to try iron and liver again.

Sept. 10, 1929: Feeling much better, flowing stopped; well enough to go back to work. Hemoglobin 65 per cent; reds 4,200,000; leukocytes 5,500; reticulated cells 8.9 per cent; polynuclears 53 per cent; lymphocytes 35 per cent; large mononuclears 10 per cent; eosinophiles 2 per cent.

Oct. 4, 1929: Feeling as well as ever. Hemoglobin 83 per cent; reds 4,740,000; leukocytes 7,200. Weight 127. Response is summarized in table 3.

TABLE 2

Date	Weight	Treatment	Hemoglobin	Red cells	Leukocytes	Reticulocytes	Catamenia
Mar. 15, '29	125	None	60%	4,490,000	7,000		
May 3, '29	126½	Fe Lactate Gr. 10	75%	5,300,000	9,600	1.4%	Nor.
May 10, '29	125½	Iron and ammonium Citrate 90 Gr.	80%	5,180,000	9,200	1.7%	Nor.
July 12, '29		"	85%				

Further Discussion

Tables 1 to 3 show in brief form the response of this patient to treatment during three separate relapses in her anemia over a period of slightly more than a year. During the first period of treatment she had one-half pound of liver daily in addition to 10 grains of ferrous lactate, and her hemoglobin rose from 40 per cent to 80 per cent in a period of six weeks. In the second period of treatment she had no liver, but took 10 grains of ferrous lactate only. During six weeks her hemoglobin rose only from 60 per cent to 75 per cent. That a larger dose of iron would have been more effective is suggested by the increase in the hemoglobin and the reticulated cells following the change to 90 grains of iron and ammonium citrate.

During the third period of treatment she was started on 90 grains of iron and ammonium

TABLE 3

Date	Weight	Treatment	Hemoglobin	Red cells	Leukocytes	Reticulocytes	Catamenia
Aug. 25	122½	None	50%	3,220,000	8,000	2.6%	Profuse
Sept. 3		Fe & Am. Citrate 90 gr.	50%			1.8%	"
Sept. 10		Fe & Liver	65%	4,200,000	5,500	8.9%	Normal
Oct. 4	127	Fe & Liver	83%	4,740,000	7,200		

citrate, and at the end of a week there had been no rise either in hemoglobin or the reticulated cells. Then raw liver was added without any other change, and within a week there was an increase in the hemoglobin from 50 per cent to 65 per cent, and in the reticulated cells from 1.8 per cent to 8.9 per cent. Possibly one week was not enough to allow for a response to large doses of iron alone, but Dr. Minot's work suggests that where iron is effective a large increase in the reticulated cells will be seen within a week. In this patient the combination of liver and iron, even when the iron is in small doses, seems to be more effective than iron alone.

Why this patient should relapse to an anemic state when she discontinues treatment is not known. As long as she continues to take either liver or iron or iron alone in large doses her hemoglobin remains normal and she has no excessive bleeding; but if she omits this treatment, even though she is on a good diet and makes no change in her manner of living, she soon begins to have menorrhagia and her hemoglobin slips down to a level of 40 to 50 per cent. It appears that she will have to continue the use of iron if she is to remain well.

DIAGNOSIS

Menorrhagia.
Secondary anemia.

A WORLD WAR AGAINST DISEASE

St. Louis, Mo., Nov. 11—Pleading for a new world war—against such enemies of society as disease, accidents, ignorance, poverty, crime, unhappiness, and other social problems—Lewis H. Carris, of New York City, managing director of the National Society for the Prevention of Blindness, speaking at an Armistice Day banquet in St. Louis, November 11, urged all nations to become allies and to refuse an Armistice to these common foes of society. This would be an excellent way to employ the fighting instinct of the human race, to which has often been attributed the recurrence of wars between men, he said.

Through the various social welfare and public health programs of the League of Nations, the League of Red Cross Societies and similar international bodies, Mr. Carris pointed out, this humanitarian war spirit has already begun. As an example, he cited the case of a "war council" which he attended at The Hague, Holland, on September 14 of this year.

"Although The Hague is noted as a world center of arbitration," Mr. Carris said, "there was no arbitration of the problem presented to this conference of September 14—the problem of what to do about the fact that a two-year study had disclosed a population of at least 2,500,000 blind people in the world, most of whom had become blind from preventable causes. The problem was met by a formal declaration of war against eye diseases and accidents by representatives of 28 nations who formed the International Association for the Prevention of Blindness in order to co-öperate in their struggle against the common enemy.

"We hope there will be no armistice to stop the war on blindness. General headquarters are for the present in Paris, and the Major Generals directing this offensive are members of an Executive Committee which includes scientists of Germany, Great Britain, Greece, Hungary, Italy, Japan, Holland, Poland, France, Switzerland, and the United States. Divisions will be formed in all parts of the world, and we hope that every country will prove an active ally.

"In the United States the National Society for the Prevention of Blindness has been engaged in a fight for conservation of vision for the last 15 years. Although some progress has been made, much more remains to be accomplished. Equally important with the question of how to prevent blindness is the problem of how to deal with the life adjustments necessary for the great number of people—several times the number of blind—whose eyesight is so poor as to form a serious vocational handicap."

How vision-handicapped soldiers were rehabilitated after the war was described by Dr. W. C. Gibson, Medical Officer in Charge, United States Veterans Hospital, Jefferson Barracks, Mo. Because the average blind man often changes from one occupation to another before definitely finding his niche, Dr. Gibson said, the Veterans Bureau assisted blind ex-service men to "find themselves again" first before giving them vocational courses.

"It was found desirable, for the blind man, after his period of prevocational training, to be returned to his home for a brief period to make necessary social and other adjustments.

"During that time it was found to be advisable to give the man a practical tryout training to determine which branch of vocational activity he was most fitted to pursue. If he thought that he wanted to take a course in poultry raising, it was deemed advisable to put him on a farm under actual working conditions so that he might know what the work required. Similarly it was deemed advisable for him to receive practical training in a store to determine whether he possessed the qualifications of success in salesmanship.

"It was recognized that some of the men would never be rehabilitated in the sense that they would become economically independent as a result of their own earnings, but it was recognized nevertheless that the majority receiving training would be enabled to earn their own living, if they so desired. The great value of the work done lies in the fact that the men were shown that blindness need not be a handicap to success."

The Armistice Day banquet was held in Hotel Chase under joint auspices of the National Society for the Prevention of Blindness, and the Lloyd R. Boutwell Post No. 136, American Legion, of St. Louis.

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DIABETIC COMA

"DIABETIC coma—a medical state, possible to prevent, expensive, time consuming, and difficult to treat, recovery a miracle to all beholders, but death a lasting blot upon the reputation of the patient or his physician."

Speaking recently in New York City at a symposium upon the prevention of chronic disease, Foster Kennedy, Professor of Neurology at Columbia, remarked that it was difficult to find a case of tabes to show the students, so efficacious had the treatment of syphilis become. It is equally heartening to read upon another page of this number of the JOURNAL* of the transformation in the frequency and in the results of treatment of diabetic coma "Then and Now". Diabetic coma should certainly be banished as a cause of death of the diabetic patient. Treatment of the condition is eminently successful and all authors, who report a series of cases, record a constantly increasing percentage of recoveries and whereas this is gratifying, we are of the belief that today more emphasis should be put upon its prevention.

*See Diabetic Coma by Reginald Fitz on Page 967.

The prevention or early recognition and treatment of diabetic coma in a doctor's practise stamps that doctor as alert, painstaking in his work, and conversant with the fundamental principles of diabetes. To detect its approach in the mild diabetes of pregnancy, to be careful enough to examine the urine of a fulminating pneumonic patient, seen for the first time, and thus diagnose diabetes and severe acidosis, to realize that appendicitis and coma in a diabetic child may be co-existent, these are achievements which reveal the physician as a practitioner of high order and sooner or later will bring him the recognition he deserves. The coma case which is saved in a hospital is the coma case early recognized in the home. Hospitals often report in their coma series only those cases of severe acidosis whose carbon dioxide combining power is 20 volumes per cent. or below; if they simultaneously reported in a separate group all cases which entered with an acidosis of less alarming proportions, as for instance between 21 and 30 volumes per cent. CO_2, they would show greater recognition of the growing diagnostic and therapeutic skill of the medical attendants who secure their admission.

Upon the shoulders of the family doctor ultimately rests the hope of the prevention of diabetes and of diabetic coma. Every opportunity therefore must be afforded him to familiarize himself with the modern treatment of diabetes. No diabetic should be discharged from a hospital without diligent effort made to return him to his own physician or to insist, if he has none, that he find one near his home. That clinic which seeks to treat its diabetics exclusively without the assistance of the family physician will not only fail to get the best results with its own patients, but we believe is derelict in its duty to the broader aspects of medicine. The information which accompanies a patient when discharged from a hospital should not only protect that patient from coma for life, but should be of such educational value to the doctor that he can utilize it in the treatment of other diabetics whom he does not feel it necessary to send to a hospital. For every diabetic day spent in a hospital, we suspect there must be a hundred diabetic days lived in the home and the shepherds who watch these diabetic sheep as they wander and stray through life are the doctors who practise alone unaided by hospital facilities. Recently we have learned that during 1928 in Massachusetts coma mortality in hospitals and outside of hospitals was essentially identical. About 9 per cent. died of coma. Possibly this figure is too low because unfortunately many certificates bear the term diabetes alone. So low a coma mortality is a splendid tribute to our State and is evidence of prophylactic coma treatment.

Success in the treatment of the patient actually in coma comes when the patient receives continuous attention until well over his acidosis.

In the words of our Author "Camp on the case" but we would add *and work too.* Many a patient will be rescued by following his clinical course with blood pressure readings, observations of the pulse and hourly analyses of the urine, even if his physician is not so fortunately placed as to be able to secure data upon the content of sugar and carbon dioxide in the blood.

SHALL RELIGIOUS SCRUPLES HAVE EFFECT IN MODIFYING PUBLIC HEALTH REQUIREMENTS?

We call attention to the letter of Dr. S. B. Woodward on another page of this issue* and request careful reading of the text so that there may be no misunderstanding of the argument. Careless reading might carry the impression that this letter is an attack on Christian Science as a religious belief. This we believe is not the purpose of the letter but rather to show that this or any other religious belief should not be employed to antagonize scientific preventive medicine. We do not believe that there need be any conflict between religion and science. Each has its own field and each must bear the test of its power to meet the needs of humanity. Certain fundamental principles underlying religious belief are common to those forms of religion which have won the approval of the larger bodies of human beings.

Certain theories based on facts have been recognized by the great proportion of persons possessing minds trained under scientific methods. Science teaches us how to live, how to profit by environment and contact and how to escape many dangers. It has shown that the human body is vulnerable to many diseases and has made plain methods by which many of these diseases may be prevented or avoided. Scientific study has been concentrated on smallpox and the consensus of public health authorities is that it is a communicable disease dangerous to individuals and a menace to the public and that it is a community problem. Furthermore that its development can be controlled almost to one hundred per cent. of the population.

Since it is dangerous to life, the most valuable possession of mankind which Society is striving to protect, where is there any sound argument against the generally accepted method of prevention.

How can any form of religious belief include antagonism to life saving methods?

Society must, as suggested in Dr. Woodward's letter, array itself against that feature of religious belief which tends to promote disease.

We do not attack Christian Science or any other religion which confines its teachings and ministrations to the spiritual and ethical aspirations of the human mind, but we do call for vigorous conflict with error. We want protection from disease for all even though in their ignorance there be some who cannot appreciate the solicitude of the medical profession.

*See Page 1012.

THE DANGER INCIDENT TO CHILD-BIRTH IN THE UNITED STATES

Dr. James Heyman of Sweden who was in attendance at the Congress of the American College of Surgeons in Chicago recently is quoted as saying that "the average woman in America takes twice as great a risk with her life in becoming a mother as the peasant woman in some remote Swedish village."

His explanation is that the Swedish Government requires of physicians special training in obstetrics. He was quoted in the daily press as saying that "obstetrical training of American medical students is sadly inadequate."

We are not ready to subscribe to this statement in so far as its application to Class A Medical Schools is concerned. While the practice of obstetrics may not be ideal in all localities there are probably other factors entering into the cause of the mortality rate in this country than medical service.

SUFFOLK DISTRICT MEDICAL SOCIETY

The attention of the local profession is invited to the talk on the treatment of acute asphyxia to be given at the next meeting of the Suffolk District Medical Society in the amphitheater of Building C, Harvard Medical School, Wednesday, November 20, at 8:15 P. M

The practicing physician is not often called upon to treat acute asphyxia, but when he is, the call is very urgent. It is of the utmost importance to every doctor to have the latest knowledge concerning what can be done in these emergencies. Most of us know in a general way what to do in cases of drowning, although our technique of artificial respiration may leave much to be desired. We may have heard that carbon dioxide and oxygen mixtures are helpful in cases of gassing by carbon monoxide, whether from automobiles or illuminating gas; but how many of us know of the remarkable results that may be obtained in cases of asphyxia due to paralysis of the respiratory apparatus caused by diseases such as acute poliomyelitis? All this will be discussed fully and the apparatus involved demonstrated by Dr. Cecil K. Drinker and Mr. Philip Drinker. We hope many doctors will avail themselves of this important opportunity.

THIS WEEK'S ISSUE

Contains articles by the following named authors:

Fitz, Reginald. A.B., M.D. Harvard 1909. Associate Professor of Medicine, Harvard Medical School. Physician to Peter Bent Brigham

Hospital. His subject is: "Diabetic Coma,—Then and Now." Page 967. Address: 721 Huntington Avenue, Boston.

BUCK, ROBERT W. A.M., M.D. Harvard 1921. Associate Chief of Medical Clinic, Boston Dispensary. His subject is: "The Diagnosis of Nephrosis." Page 973. Address: 475 Commonwealth Avenue, Boston.

BLOODGOOD, JOSEPH C. B.S., M.D. University of Pennsylvania 1891. F.A.C.S. Resident Surgeon, Johns Hopkins Hospital, 1893-1897. Associate in Surgery, Johns Hopkins Hospital and University, 1897-1903. Associate Professor of Surgery, Johns Hopkins Hospital and University since 1903. Chief Surgeon, St. Agnes Hospital, Baltimore. His subject is: "Codman's Registration of Bone Sarcoma and Its Bearing Upon A Study of Malignancy Elsewhere." Page 979. Address: 904 North Charles Street, Baltimore, Maryland.

COTTON, FREDERIC J. A.B., A.M., M.D. Harvard 1894. F.A.C.S. Surgeon, Boston City Hospital. Chief of Bone and Joint Service, Boston City Hospital. Address: 520 Commonwealth Avenue, Boston. He writes on: "Non-Union of Fractures of the Long Bones," page 982 and "Osteomyelitis with Special Reference to Treatment," page 986. Associated with him in this issue is:

BERG, RICHARD K. B.S., M.D. University of Oregon Medical School, 1926. Formerly Interne, Saint Vincent's Hospital, Portland, Oregon and the Orthopedic Service, Carney Hospital. Also resident, Boston City Hospital, Sixth Surgical Bone and Joint Service. Now Gibney Fellow at the Hospital for Ruptured and Crippled, New York. Address: Ruptured and Crippled Hospital, 321 E. 42nd Street, New York City. They write on: "Two-Stage Pedicled Graft to Replace Unsatisfactory Scar", page 981; "Raynaud's Disease and the Trauma of Occupation", page 985; and "'Fender Fracture' of the Tibia at the Knee", page 989.

TRUESDALE, PHILEMON E. M.D. Harvard 1898. F.A.C.S. Surgeon to the Truesdale Hospital and Earle P. Charlton Surgery. His subject is: "Medical Illustrations of Antiquity to the Medical Audio-cinema of the Present." Page 996. Address: 151 Rock Street, Fall River, Mass.

ARLINGHAUS, F. H. Mechanical Engineer, Stevens Institute of Technology, 1924. Representative, Department of Educational Talking Pictures, Electrical Research Products, Inc. Subsidiary, Western Electric Company. He presents an "Address to the New England Surgical Society." Page 999. Address: Electrical Research Products, Inc., 250 West 57th Street, New York City.

MISCELLANY

DR. C. C. LITTLE'S QUALIFICATIONS FOR THE POSITION OF DIRECTOR OF THE AMERICAN SOCIETY FOR THE CONTROL OF CANCER.

Dr. Clarence Cook Little, formerly President of the University of Michigan and now in charge of the Roscoe B. Jackson Memorial Laboratory for Cancer Research at Bar Harbor, Maine, has been appointed Managing Director of the American Society for the Control of Cancer. The choice of Dr. Little for this important position was made by the Executive Committee of the Society, representing a board of thirty directors, which includes the leading cancer experts of the country.

Dr. Little brings unusual qualifications to his new and difficult task of directing the activities of the leading organization in the world devoted to the advancement of the knowledge and control of cancer, which now contests with tuberculosis the distinction of being the chief cause of death.

Dr. Little's experience as an educator and a man of affairs fully qualifies him to direct the work of the Society in the field of public education and economics. His attainment and contributions in scientific research, especially on the question of heredity, enable him to speak with authority on the scientific aspects of the cancer question. His appointment is therefore a matter of congratulation both to the Society and to the public.

Dr. Little was born in Brookline, Massachusetts, on October 6, 1888, and was educated at Harvard where he received the degree of A.B. in 1910, M.S. in 1912 and D.Sc. in 1914. He took an important part in undergraduate activities and was captain of the varsity track team. During and following his graduate training he served Harvard University, first as research assistant in genetics, then as research fellow and finally as associate in comparative pathology. Following the war he joined the staff of the Station for Experimental Evolution of the Carnegie Institute in Washington. Among his scientific associations are the New York Academy of Medicine, the American Society of Naturalists, American Society of Zoölogists, and the Eugenic Research Association.

In addition to his scientific career Dr. Little has held several important administrative posts in the educational field. Notable among them have been Secretary of the Harvard Corporation, Assistant Dean of Harvard College, President of the University of Maine and President of the University of Michigan.

MEDICAL SUPERVISION OF WOMEN WORKERS

Because of evidence of certain hazards in some occupations in which women are employed the Consumers League of Massachusetts has conducted a survey and has found that workers in some kinds of candy, rubber, boot and shoe and tanning industries are exposed to poisons.

Six cases of benzol poisoning resulting in three deaths were reported in Massachusetts in 1927; in addition to benzol, methyl alcohol and amyl acetate

are used in some industries where women are employed and are known to be injurious to those exposed. Even chocolate dippers may develop dermatitis and other disturbances incident to the low temperature in the chocolate rooms. Naphtha rubber cement is deleterious to those exposed to the vapor. The list might be extended but the fact remains that women are suffering from a variety of irritants and poisons which seriously affect health. This indicates the necessity of careful physical examinations from time to time and control of the processes so that the health of workers will not be impaired. The League recommends the adoption of suitable codes regulating the use of these poisons, the appointment of an industrial health inspector and industrial clinics in hospitals and dispensaries and research in the field of industrial hygiene.

A TUBERCULOSIS CLINIC IN SPRINGFIELD

Dr. Paul Wakefield of the Massachusetts Tuberculosis Commission has been in conference with the principals of the public schools of Springfield, Massachusetts regarding the ten-year tuberculosis study in Massachusetts. Cases in that region will be studied in a clinic devoted to the early diagnosis of the disease before it invades the lungs. Dr. Wakefield will spend several weeks in conference with principals of the elementary schools. The information conveyed to these clinics will be extended to those who will carry on the campaign.

RECONSTRUCTION OF THE TEMPLE UNIVERSITY MEDICAL SCHOOL

Plans have just been completed and ground broken in Philadelphia for what promises to become an example of an up-to-date medical school.

This imposing structure, to be erected at Broad and Ontario streets, opposite the Samaritan Hospital, for the School of Medicine of Temple University, will involve an approximate expenditure of $1,500,000, and, when completed next year, will be probably a perfectly equipped medical educational centre.

William H. Lee, architect of the proposed "Temple of Learning," or skyscraper building group to be erected for Temple University, is the designer of the building. Already the foundations are well under way. The new building will replace the present congested headquarters of Temple's School of Medicine.

With an adaptation of Georgian architecture, to conform to the general design of the Samaritan Hospital of Temple University, the building will cover a lot 100 by 198 feet, with the exception of a ten foot court extending around the west and north sides.

The building will be built in the shape of an "E" with two courts facing the West. The first two floors will be occupied by an Out-Patient Department for the Samaritan Hospital. The upper five floors and the basement will be used for the purposes of the Medical School.

The exterior of the building will be of Colonial red brick and limestone. The construction is designed for reinforced concrete throughout with hollow tile partitions. The Out-Patient Department will be entered at the corner of Broad and Ontario streets. A large waiting room for Dispensary patients will be located just off the main entrance to the Dispensary. This waiting room will communicate with the various Departments by a wide corridor and by stairway and elevator to the dispensary on the second floor. Each department is provided with an adequate waiting room and each department is designed and equipped in the most modern manner to take care of dispensary work.

The entrance to the Medical School will be from the center of the building on Broad street. Communicating with the lobby inside the entrance will be the administrative part of the Medical School, the elevators and the library. The third floor will contain the Department of Pharmacology and an auditorium, with a sufficient capacity to accommodate the entire school. This auditorium will be susceptible of division into two large lecture rooms. About one third of the third floor is left unfinished for future expansion of Medical School or dispensary.

The fourth floor will be given over entirely to the Departments of Chemistry and Physiology. Each department will have a laboratory large enough to accommodate a class of 100 students. Each of these departments will also be provided with lecture rooms to accommodate 100 students each; the balance of the floor being devoted to research laboratories and staff offices.

The fifth floor will be occupied by the Pathology and Bacteriology Departments. It will also contain two large laboratories, a number of smaller research laboratories, staff offices and special rooms of various character. On this floor also will be provided a large museum of morbid anatomy.

The sixth floor will be devoted entirely to the Department of Anatomy. There will be in the north wing a dissecting room to accommodate 100 students. In the south wing will be provided a large lecture room, conference rooms and museum. The center wing will be for operative surgery and in this wing will also be located the mortuary refrigerator.

The balance of the floor will be used for research laboratories and staff offices.

On the seventh floor will be the animal storage and Medical and Surgical Research Departments. The basement will be occupied by students' locker rooms and lounging rooms, library book stacks, boiler plant and for various storage purposes. A mortuary incinerator will be installed in the basement adjoining the boiler room.

The equipment for the various departments for the School and the dispensary is now being designed and will be placed under contract in the near future. It is expected that the building will be complete and equipped ready for occupancy by September, 1930.

Simultaneously, Dr. Charles E. Beury, president of Temple University, has announced the creation of six new departments in the School of Medicine. They are medical research, neuro-surgery, physical therapy, neurology, bronchoscopy and otology.

SMALLPOX INCREASING

The United States Public Health Service reports that "smallpox is sharply increasing in prevalence."

COMPARISON OF DISEASE INCIDENCE IN CONNECTICUT WITH 1928 AND SEVEN YEAR AVERAGE MONTH ENDING OCTOBER 26, 1929

DISEASES	1929					1928			
	Wk. ending Oct. 5	Wk. ending Oct. 12	Wk. ending Oct. 19	Wk. ending Oct. 26	Average cases reported for week corresponding to Oct. 26 for past seven years.	Wk. ending Oct. 6	Wk. ending Oct. 13	Wk. ending Oct. 20	Wk. ending Oct. 27
Cerebrospinal Men.	-	-	-	1	-	-	-	4	-
Chickenpox	27	23	54	82	52	15	47	38	59
Conjunctivitis Inf.	-	-	-	-	-	-	1	-	-
Diphtheria	14	15	36	24	41	29	21	19	18
Dysentery Bacillary	-	-	-	-	-	-	-	1	-
Encephalitis Epid.	-	3	-	-	-	-	-	1	-
German Measles	4	2	1	2	1	3	1	5	1
Influenza	2	34	4	3	3	12	3	2	3
Malaria	1	-	-	-	-	1	-	-	-
Measles	4	-	3	2	40	5	27	35	48
Mumps	12	11	15	12	16	7	12	9	18
Paratyphoid Fever	6	2	1	-	-	-	-	1	-
Pneumonia (Broncho)*	11	7	15	21	13	23	9	26	16
Pneumonia (Lobar)	13	12	16	25	21	16	28	18	19
Poliomyelitis	1	-	2	1	3	6	4	3	6
Scarlet Fever	14	22	24	33	45	12	22	21	22
Septic Sore Throat	1	2	3	1	12	3	-	1	3
Trichinosis	1	-	-	-	-	1	-	-	-
Tuberculosis (Pul.)	33	18	18	31	28	43	25	20	16
Tuberculosis (O.F.)	1	1	1	5	2	4	1	1	1
Typhoid Fever	4	2	4	3	3	1	6	1	1
Whooping Cough	25	24	37	36	44	43	27	33	35
Gonorrhoea	30	26	37	33	28	47	32	49	60
Syphilis	30	19	37	28	25	54	35	61	48

* Made reportable January 1, 1925.

REMARKS: No cases of Asiatic cholera, Glanders, plague, rabies in humans and Yellow fever during the past seven years.

DR. J. C. McCANN WILL BE A MEMBER OF THE FALLON CLINIC

Dr. James C. McCann, a graduate of the Harvard Medical School in 1924, formerly an intern at the Carney Hospital and Fellow in Surgery with the Mayo Foundation, Rochester, Minnesota, will be associated with the Fallon Clinic in Worcester, Massachusetts. He has had articles published in the *American Journal of Physiology* and the *Archives of Surgery* dealing with the physiology and chemistry of the stomach and with the experimental peptic ulcer.

DISCUSSION OF SPAHLINGER TREATMENT RULED OUT OF ORDER

The President of the Executive Grand Council of the Geneva Municipal Government declined to permit discussion of the Spahlinger treatment of tuberculosis, although there was a strong sentiment in favor of consideration of the value of the assertions made by Spahlinger, who, although not a physician, makes claims of cures of tuberculosis by his serum. Eleven Swiss physicians appointed by the local government have declared this alleged discovery to be a "grandiose fraud."

Spahlinger is reported to be treating many American and British patients.

THE SCARLET FEVER MENACE IN JAMAICA PLAIN

Dr. John A. Ceconi addressed a meeting of parents in the Francis Parkman School District, Jamaica Plain, recently and urged co-öperation of parents and doctors in dealing with the cases of scarlet fever in that section. Most of the cases have been mild and one purpose of the meeting was to dispel any undue fear among the parents and children.

INFLUENZA SEASON APPROACHING

Assistant Surgeon R. C. Williams, United States Public Health Service, has stated that beginning in November and continuing through March influenza is found to reach its seasonal prevalence.

The observance of well-known rules of hygiene are of importance in preventing unusual numbers of cases, especially avoiding mingling with crowds and living as much as possible in the open.

CORRESPONDENCE

SHALL RELIGIOUS SCRUPLES HAVE EFFECT IN MODIFYING PUBLIC HEALTH REQUIREMENTS?

Mr. Editor:

The *Christian Science Monitor* in a recent issue calls attention to the fact that the full bench of the Massachusetts Supreme Court upheld a Superior Court verdict that Howard Green of Pittsfield was guilty of failure to send his children to a school from which they had been debarred because they were unvaccinated. The decision points out that whatever Mr. Green's religious scruples may be as presented in his defense the letter of the law is clear, that vaccination is a condition precedent to the right of a child to attend public school and that Mr. Green by his own act kept the children away. Mr. Green's quotation from the State Constitution on the subject of religious liberty was declared to be irrelevant.

It is well known to those familiar with vaccination legislation that its opponents come largely from followers of Christian Science. Never, however, until this year have religious scruples been openly put forward as a reason for non-compliance with either the present law or a proposed modification which would treat all educational institutions alike. This year, however, opponents of vaccination were conspicuously absent from the hearing before the legislative committee on public health, there was no opposition when the matter was brought before the legislative body, and it was only after the Senate as well as the House had apparently finished with the subject that a body of citizens went to Governor Allen with the suggestion that the statement of objection on account of religious scruples should allow unvaccinated children to attend public as well as private schools. The whole matter was then referred to the next legislature.

It seems to me proper at this time to say that if religious scruples are to be allowed to nullify laws enacted to protect the public health, for which purpose and for which purpose only are public school children vaccinated, the logical outcome is that all sanitary laws can be made equally null and void. A statement that religious scruples prevent the destruction of animal life (the belief of millions in India) would send to school children with heads swarming with lice, and every attempt to improve conditions in the home, the workshop and the school *could* be entirely thwarted.

Science and religion have been at war since the beginning of time, but I had supposed that in this age and generation, and particularly in this America and this Massachusetts no intelligent person would suggest that science was to be cast aside simply on account of religious belief. I am glad that the matter has at last come out into the open. I am glad that pseudo scientific objections to vaccination are pushed into the background, and that those who are trying so valiantly to hold back the wheels of progress are willing to simply say "my religion prevents me from accepting what medical science has so painfully learned."

Were the disastrous effects of non-compliance to be confined to those offering religion as a barrier to proper sanitary procedures, we might possibly allow them to themselves reap the reward of their own folly, but to permit them to offer innocent children "to Moloch" and expose those not agreeing with them, and those the vast majority of us all, to the effects of contagious disease is too much to expect.

We must continue to fight. We must not cease our efforts until at the very least school children shall be protected by law.

Incidentally to date there have been reported this year to the State Department of Health 273 cases of smallpox. In the previous 15 years but 478 were brought to the attention of the Commissioner of Health and from January, 1914, to the first of last January, 16 years, but 321.

We have this year not only to try and improve our present vaccination law, but we must as well prevent the suggested modification of the law under which we have for so many years lived. Religious scruples as a reason for exemption from compulsory vaccination would tear away every protection, riddle with holes the restraining barrier and render ridiculous the school law and every other health law to which it was applied.

SAMUEL B. WOODWARD.

AN EXPRESSION OF APPRECIATION

To the Editor:

Through the columns of your Journal, may I express my appreciation of the treatment which was given to me by the Physicians Liability Insurance Company in the case of tort against me which has just been in the courts. No pain or effort was spared by the officers in their consideration of the case, and I wish to acknowledge this, and the thorough preparation made by the attorneys and the efficiency with which they conducted this case throughout. In these legal proceedings in which we medical men must rely completely on the experience of experts in the management of these cases, it is difficult to express adequately the relief that comes from this confidence, and the knowledge that these cases will have this thorough, efficient and skillful attention, and it is fitting that we who have had the advantage of this

protection should make known our experience for the benefit of other brother practitioners who may be obliged to go through the same experience.

E. G. BRACKETT.

166 Newbury Street.

ADDITIONS TO THE PERSONNEL OF THE STAFF OF THE MASSACHUSETTS PUBLIC HEALTH DEPARTMENT

The Commonwealth of Massachusetts
Department of Public Health
State House, Boston

November 2, 1929.

Editor, New England Journal of Medicine:

May I through your columns announce important additions to our staff.

Dr. Charles B. Mack has been appointed as District Health Officer in the Boston Metropolitan Area. He graduated from Jefferson Medical School, received a degree of Doctor in Public Health from the University of Pennsylvania, and did post graduate work in public health at Vienna and the London School of Tropical Medicine. During the war he had extensive medical experience in Serbia and elsewhere. He has had a number of hospital administrative positions and served for some time on the field staff of the College of Surgeons. Recently he has been with the American Public Health Association. He succeeds Dr. Lane, who has been promoted to the position of Assistant Director of the Division of Communicable Diseases.

Dr. Alton S. Pope has been appointed as Epidemiologist in the Division of Tuberculosis. After graduating from Tufts, Dr. Pope studied at the Harvard School of Public Health, during which time he made an extensive study of scarlet fever in Providence. For some time he was in Dr. Haven Emerson's department at Columbia, working on epidemiological problems connected with degenerative diseases. For some four years he has been Director of the Division of Communicable Disease of the Health Department of Chicago and has done work in Professor Jordan's Department at the University of Chicago. He is joining us to institute a study of the extensive material on tuberculosis, going back as far as the middle of the last century. From death returns, morbidity reports, hospital records, the records of the Chadwick Clinics, and the like, we anticipate that we can learn much about what has happened to tuberculosis in the last seventy-five years. On this we can base sound plans for future development in the control of this disease. Dr. Pope would seem to be unusually well qualified for this intricate study.

We are appointing Miss Mary P. Billmeyer as Department Consultant in Public Health Nursing. She was trained at the Presbyterian Hospital in Chicago and has done post graduate work in Chicago and Oregon. She has worked with the Red Cross in Michigan, with the Multnomah County Public Health Association in Oregon, and more recently in the Judson Health Center in New York City. She will work not only in an advisory capacity within the Department but, far more important, in such capacity to public and private agencies employing public health nurses throughout the State. In view of the fact that Massachusetts has more public health nurses employed than any other state we have felt the need of this position for a long time.

Yours truly,
GEORGE H. BIGELOW, M.D.,
Commissioner of Public Health.

BOSTON TRAFFIC COMMISSION GIVES NO PRIVILEGES TO PHYSICIANS

October 31, 1929.

Mr. William A. Fisher,
Commissioner of Traffic,
Police Headquarters,
Berkeley Street,
Boston, Mass.

Dear Mr. Commissioner:

On behalf of the physicians practicing in Massachusetts I would respectfully request that you consider the advisability of granting to them the privilege of temporary parking of their automobiles in restricted areas when they have been called to those areas to respond to an emergency professional call.

Surgical, obstetrical, and medical emergencies demand immediate attention. Specific cases can be cited where lives have been placed in jeopardy by the strict enforcement of the non-parking regulations.

Very truly yours,
R. B. GREENOUGH,
President Massachusetts Medical Society.

Boston
Traffic Commission
154 Berkeley Street

November 1, 1929.

Robert B. Greenough, Pres.,
The Massachusetts Medical Society,
165 Newbury St., Boston.

Dear Sir:

I received your letter of October 31 requesting special privileges for physicians.

This is a matter which has been seriously considered by all traffic authorities and no solution has yet been devised for this very undesirable condition.

The Courts have consistently held that we cannot grant special privileges in the public streets. They refuse to recognize special permits of this nature.

It is not only physicians who are on errands involving life and death, we have similar requests from various concerns and individuals who have a similar urgent need to use the streets in violation of the traffic rules.

I am sorry that I am unable to submit a solution of your problem which I think is deserving of a solution.

Very truly yours,
WILLIAM A. FISHER, *Commissioner.*

A PROTEST AGAINST A BOOK REVIEW

To the Editor, NEW ENGLAND JOURNAL OF MEDICINE,

Dear Sir:

A review of my book, "What Everyone Ought to Know" appeared in the JOURNAL of August 29, 1929.

Without consulting me, the last part of the review embodied a letter from Professor Prescott. This letter was in reference to my statement that Professor Prescott had received $40,000 from the Coffee Trade Publicity Committee for a research on coffee. In his letter which was printed in the review of my book he states "If the rest of the book is based on as good authority as his statement about my report I am sure it was misnamed." This is an innuendo that not only attacks me but hurts the sale of my book. He also says that the sum paid for this research was "considerably less than $40,000." As a matter of fact, the phrase "A $40,000 research on coffee by Professor Samuel C. Prescott, of the Massachusetts Institute of Technology" was published all over the United States by the Coffee Publicity Committee. To my knowledge Professor Prescott never made a statement that this price was incorrect. Therefore I had a perfect right to quote this price in my book.

Professor Prescott also states in this letter that his report of this research "distinctly stated that for a small percentage of people coffee is unquestionably undesirable." To my knowledge, as far as any advertisement that I ever saw, his restrictions on the use of coffee were never published by the Coffee Publicity Committee, nor to my knowledge did Professor Prescott object to his restrictions on the use of coffee being omitted from the advertisements of the Coffee Publicity Committee.

Also it should be noted that I distinctly state in my book, "The part of this research utilized by the coffee interests for advertising purposes stated that coffee was practically harmless." Professor Prescott apparently did not note that I did not say that he did not have restrictions somewhere in the body of his report.

In other words, I have been unjustly accused of making a false statement in my book. It is only fair to me that this letter be published in the JOURNAL in which Professor Prescott's letter taking exception to statements in my book was printed.

OLIVER T. OSBORNE.

RECENT DEATHS

MILLER—The JOURNAL records with regret the sudden death from heart disease at his home in Worcester of Lester Colwell Miller, Vice-President of the Massachusetts Medical Society and until recently dean of the medical staff of the Memorial Hospital. Dr. Miller had been ill for a month but the end came unexpectedly on November 7, 1929.

The son of Mr. and Mrs. George A. Miller, he was born in Rockport, Maine, May 15, 1867. After attending the local schools he was a student at Colby University and in 1920 received the Honorary A.M. from that institution. Entering Harvard Medical School he was graduated with the class of 1895, spent two years in study abroad and settled in Worcester, where he was visiting physician to the Worcester Isolation Hospital and to the Washburn Memorial Hospital Out-Patient Department. He rose through the grades until he was dean of the medical staff of the Memorial Hospital, resigning only two years ago to devote his time to internal medicine. Recently he had been chairman of the Medical Section of the Massachusetts Medical Society and last June was elected Vice-President of the society. He was a Fellow of the American Medical Association, a member of the New England Pediatric Society and of the Practitioners Club.

Dr. Miller was a member of several social and fraternal organizations, including the Tatnuck Country Club, Bohemian Club, Economic Club and Montacute Lodge of A.F. and A.M. His hobby was painting in oils but he did not exhibit his works in public. He had a host of friends both in the profession and out and will be sorely missed.

He is survived by his widow, who was Miss Delia Burgess. They had no children.

LIBBY—DR. EDWARD NORTON LIBBY, Professor of Theory and Practice of Medicine in Tufts College Medical School, died following a cerebral hemorrhage at his home in Jamaica Plain, November 6, 1929, aged 61.

NEWS ITEMS

ALVARENGA PRIZE OF THE COLLEGE OF PHYSICIANS OF PHILADELPHIA—The College of Physicians of Philadelphia announces that the next award of the Alvarenga Prize, being the income for one year of the bequest of the late Senor Alvarenga, and amounting to about Three Hundred Dollars, will be made on July 14, 1930, provided that an essay deemed by the Committee of Award to be worthy of the Prize shall have been offered.

An essay intended for competition may be upon any subject in Medicine, but must be accompanied by a written assurance from the author that it has not appeared previously in print, either in whole or in part, in any form, and has not been presented elsewhere in competition for a prize. The essay should represent an addition to the knowledge and understanding of the subject based either upon original or literary research. It must be typewritten, and in English acceptable for publication without necessity for editing by the Committee. Any illustrations should be appropriate and correctly annotated with the text. Essays must be received by the Secretary of the College on or before May 1, 1930.

Each essay must be sent without signature, but must be plainly marked with a motto and be accompanied by a sealed envelope having on its outside the motto of the paper and within the name and address of the author.

It is a condition of competition that the successful essay or a copy of it shall remain in possession of the College; and that it may be published by the author with the consent of the College; other essays will be returned upon application within three months after the award.

The Alvarenga Prize for 1929 has been awarded to Dr. George M. Dorrance, Philadelphia, Pa., for his Essay entitled: "Congenital Insufficiency of the Palate."

JOHN H. GIRVIN, *Secretary*,
19 South 22d Street, Philadelphia, Pa., U. S. A.

THE USE OF BACTERIOPHAGE—Equipped with a brand new weapon against disease, scientists have failed to get the hoped-for results with it because they have not known exactly how to use it. Bacteriophage, the potent germ killer, discovered by the French-Canadian, Dr. F. d'Herelle, now a Yale professor, gave promise of being the world's greatest disease-conqueror. It has fallen short of fulfilling this promise because the men who had to use it have not understood it well enough to get uniformly good results.

The age of the material, the method of administration and the amount of the dosage are points that must be settled in order to get the most successful results with this new weapon, one of its advocates, Dr. N. W. Larkum, of the Michigan Department of Health, told a group of fellow scientific men, at a meeting held in Indianapolis on October 18. The group to which he spoke was composed of men who have devoted themselves to the study of disease germs and means of combatting them. This group is an informal organization, without any name, which meets several times a year in order to exchange views and notes of progress on the members' work.

Bacteriophage kills germs in a test-tube and actually dissolves them. The hope of its discoverer and sponsors is that it will do the same thing to germs in the human body. In some cases it has killed the germs that were attacking the body, in others it has failed to do so. Dr. Larkum believes that the failures occur when the phage is used incorrectly, through ignorance of the best conditions for it to do its work.

Possibly the phage is potent only at a certain stage of its development. Up to now, the age of the material has not been given any consideration. The method of administration has been chosen with a view to getting the stuff into contact with the bacteria. Dr. Larkum suggested that it may be better to bring the material into contact with the susceptible tissues of the body, in order to make them immune to attacks of the disease germs and thus check the spread of the germs through the body. The matter of how large a dose to give also needs to be determined scientifically. "At present the selection of the dosage is entirely arbitrary, based upon some success attending a given dose. It is entirely logical to conclude that as many failures have been due to excessive dosage as have been due to an insufficient amount. Until further light has been shed upon these various factors in bacteriophage therapy, one is scarcely justified in condemning the method."—*Science.*

DR. ARTHUR L. CHUTE WILL DISCUSS A PAPER BY DR. EDWARD L. KEYES—Dr. Arthur L. Chute will attend the meeting of the Section of Genito-Urinary Surgery of the New York Academy of Medicine, November 20, and discuss a paper by Dr. Edward L. Keyes on "Prospects of the Prostate".

DR. FRANK R. OBER WILL DELIVER A LECTURE BEFORE THE NEW YORK ACADEMY OF MEDICINE—On Friday afternoon, November 22, Dr. Frank R. Ober will deliver a lecture before the New York Academy of Medicine under the title "The Relation of Orthopedics to Pediatrics".

NOTICES

A REVIVAL OF THE HARVARD EXTENSION COURSES IN WORCESTER

Announcement has been made by Dr. Ernest L. Hunt and Dr. William F. Lynch of the renewal of the Harvard extension courses in Worcester which were so popular several years ago.

The first lecture was given by Dr. James P. O'Hare, November 6. Dates of the subsequent lectures are as follows:

November 20, 7:30—"Varieties of Edema and Their Treatment," Dr. Henry A. Christian, Peter Bent Brigham Hospital, Boston.

November 27, 8:15—"Coronary Artery Disease," Dr. Samuel A. Levine, 270 Commonwealth Avenue, Boston.

December 4, 8:15—"Dietary Anemia, Eczema," Dr. Lewis W. Hill, 319 Longwood Avenue, Boston.

December 18, 8:15—"Immunity and the Use of Serums and Vaccines," Dr. Benjamin White, 375 South Street, Jamaica Plain.

January 15, 8:15—"Treatment of Skin Diseases," Dr. C. Guy Lane, 416 Marlborough Street, Boston.

January 22, 8:15—"Advances in Knowledge of Endocrines," Dr. Joseph C. Aub, Huntington Hospital, Boston.

This is an unusual opportunity for instruction for a very small sum.

REMOVAL

Dr. Gerald Norton Hoeffel announces the removal of his office from 270 Commonwealth Avenue to The Longwood Medical Building, 319 Longwood Avenue, Boston.

UNITED STATES CIVIL SERVICE EXAMINATION

The United States Civil Service Commission announces the following open competitive examination:

Assistant Medical Officer (Dermatology)

Applications for assistant medical officer (dermatology) must be on file with the Civil Service Commission at Washington, D. C., not later than December 4.

The examination is to fill a vacancy in the U. S. Public Health Service, Ellis Island, New York.

The entrance salary is $3,000 a year.

The duties are to diagnose skin diseases in inspection of passengers and crews of vessels; and to perform related duties as assigned.

Applicants must have at least one year of postgraduate training or specialized experience in dermatology.

Full information may be obtained from the United States Civil Service Commission at Washington, D. C., or the secretary of the United States Civil Service Board of Examiners at the post office or customhouse in any city.

REPORTS AND NOTICES OF MEETINGS

THE PLYMOUTH COUNTY MEDICAL SOCIETY

Before a large audience that filled Massasoit Hall, Tuesday evening, October 29, 1929, Dr. Chevalier Jackson of Philadelphia addressed perhaps one of the greatest number of medical men that has ever convened in the history of Plymouth County. The Plymouth District Medical Society entertained Dr. Jackson by a reception committee consisting of Dr. Thomas H. McCarthy, President of the Society; Dr. John F. Callahan, a former student of Dr. Chevalier Jackson, who was chairman of the Reception Committee, Dr. David B. Tuholski, Dr. John J. McNamara, Dr. Charles G. Miles, Dr. W. H. Pulsifer, Dr. F. W. Murdock, Dr. Richard B. Rand, Dr. Alfred C. Smith and Dr. John P. Shaw. Physicians and surgeons were present from many cities and towns of Massachusetts, and also from Rhode Island and New Hampshire. The magnetic attraction of this great Bronchoscopist was demonstrated by the tremendous audience that gathered to pay him homage. Dr. John F. Callahan was mainly responsible for his presence in Brockton. In introducing Dr. Chevalier Jackson, Dr. Thomas H. McCarthy, President of the Plymouth District Medical Society, said "The world at large has its Edison, its Marconi and its Mme. Curie, but we feel that Dr. Chevalier Jackson who has perfected his line of work in the removal of foreign bodies from the lungs can be classed with these three great discoverers, and at the present time is the most eminent man in the world in his field of practice. When as a young man Dr. Jackson began this work, he practised on dogs, cats and chickens, making instruments with which to perform his operations, and finally developed the technic which has made him an expert in his field. His students are now located throughout the earth, performing operations which they learned under the direction of Dr. Chevalier Jackson, our guest, the speaker of this evening."

He then introduced Dr. Chevalier Jackson, who took for his subject, "Bronchoscopy as an Aid to the Internist and Surgeon in the Diagnosis and Treatment of Diseases of the Lung." He illustrated his talk with the aid of lantern slides, moving pictures, and also chalk talks, drawing some very beautiful pictures illustrating the different points that he was discussing. He showed a large chart representing hundreds of small foreign bodies that he had removed during his work in the air and food passages. Dr. Jackson stated that when a patient has choking, gagging and wheezing, often followed by a symptomless interval, it is usually one of the initial symptoms of a foreign body in the air or food passage. It may be in the pharynx, larynx, trachea, bronchi or nasal chamber, or even in the tonsil. In the food passage, it may be in the esophagus, stomach, or may have been passed by the bowel or might have been coughed out without the knowledge of the patient. By far the greatest number of foreign bodies are the chicken bones, and most of which have been removed from women patients.

When you get a foreign body in the larynx, it is usually characterized by a croupy cough, wheezing, hoarseness, sometimes hemoptysis and cyanosis. The obstructing foreign body causes sudden death by laryngeal impactions. Direct laryngoscopy for diagnosis is indicated in every child having laryngeal diphtheria without faucial membrane. In the presence of laryngeal symptoms, always think of the following, a foreign body in the larynx or one loose or fixed in the trachea, digital displacement due to efforts at removal, instrumentation, overflow of food into the larynx from esophageal obstruction due foreign body. Laryngeal symptoms may persist because of trauma of a foreign body which may have passed into the air or food passages. Roentgen ray, laryngoscopy, direct in children, indirect in adults, without anesthesia general or local, is the only way to make a laryngeal diagnosis. Before doing a diagnostic laryngoscopy, general preparation should be made for taking a swab specimen and also for bronchoscopy and esophagoscopy.

Dr. Jackson went into details in regard to the bronchial and trachial foreign bodies. He said that the audible slap, palpatory thud and asthmatoid wheeze is pathognomonic. He said that cough, dyspnea, and cyanosis are often present. Diagnosis is by Roentgen ray, auscultation, palpation and bronchoscopy. Listen long for audible slap. It is best heard at the open mouth during a cough. The asthmatoid wheeze is heard with the ear or stethoscope bell at the patient's open mouth. In the bronchial foreign body, he said that the initial symptoms were coughing, choking, the asthmatoid wheeze and hoarseness and dyspnea are often present. At this point in his lecture he showed a picture of a patient that apparently had swallowed a tooth. He stated that bones and metallic bodies after months or years brought about changes which caused chills, fever, sweats, emaciation, clubbed fingers, incurved nails, cough, foul expectoration and hemoptysis, in fact all of the symptoms of chronic pulmonary sepsis, also signs which may suggest pulmonary tuberculosis but the apices are normal and tuberculous bacilli are absent from the sputum.

Vegetable organic foreign bodies, such as peanut kernels, beans, watermelon seeds, peas, cause at once violent laryngotracheo-bronchitis with toxemia cough and fever. The gravity and severity is inversely to the age of the child. The Roentgen ray is the most valuable diagnostic means that we have but careful notation of physical signs by an expert should be made in all cases. Expert ray work will show all metallic foreign bodies and many of less density such as teeth bone, shell, buttons, etc. If the ray is negative for foreign body but indicates bronchial obstruction a diagnostic bronchoscopy should be made. Peanut kernels and watermelon seeds in the bronchi produce obstructive emphysema of the invaded side. Fluoroscopy shows the diaphragm flattened, depressed and of less excursion on the invaded side. At the end of expiration, the heart and mediastinal wall move over toward the uninvaded side and the invaded lung becomes less dense than the uninvaded lung from the trapping

of the air by the expiratory valvelike effect of obliteration of the forceps spaces that during inspiration afford air ingress between the foreign body and the swollen bronchial wall. This partial obstruction causes obstructive emphysema which must be distinguished from compensatory emphysema in which the ballooning is in the unobstructed lung because its fellow is wholly out of function, through complete blocking of the main bronchus of the invaded side.

Dr. Jackson stated that in the case of an esophageal foreign body after initial choking and gagging, or without these, there may be a subjective sense of a foreign body, constant or more often on swallowing. Odynophagia and dysphagia may or may not be present. Hematemesis and fever may occur from the presence of the body or rough instrumentation. Symptoms may be due to the overflow of secretions or attempts to swallow through the obstructed esophagus, or to the erosion of the foreign body through the esophagus into the trachea, or to trauma inflicted on the larynx during attempts at removal, digital or instrumental, though the foreign body is present. Diagnosis is made by Roentgen ray first without, then if necessary with a capsule filled with an opaque mixture. Flat objects like coins always lie with their greatest diameter in the coronal plane of the body when in the esophagus. In the saggital plane when in the trachea. Lateral anteroposterior and sometimes also quartering Roentgenograms are necessary. One taken laterally, low down on the neck, but clear of the shoulder, will often show a bone invisible in the anteroposterior exposure.

The moving pictures seemed to be the most interesting part of Dr. Jackson's lecture because he thereby illustrated the use of the bronchoscope in removing foreign bodies and in inducing respiration where the lungs had collapsed and the passages had been clogged. Illustrations of young children were shown on the operating table with the bronchoscopist and his team at work, without the aid of an anesthetic, where the children after the operation would slide from the operating table and walk quickly away. One of the pictures shown was a little colored boy who was suffering from tuberculosis. A large abscess had formed on the left side. Tracing the source of the trouble, it was found that the boy had swallowed a quarter and it had become lodged in the child's bronchi and it had been there for five years. Aspiration had been performed and the foreign body located.

Dr. Jackson showed unlimited knowledge of his subject. He talked very slowly and steadily, and even those who were not familiar with some of the points that he tried to bring out were enthusiastic in speaking of the thorough way in which he defined his subject. At the end of his lecture and demonstration, Dr. Jackson was given thunderous applause. He then retired.

At the conclusion of the lecture by Dr. Jackson, a limited discussion followed. Dr. Leroy A. Schall, of the Boston City Hospital, discussed the subject by the aid of several lantern slides. Dr. Schall was very thorough and showed a full knowledge of his subject. He was very well received. Dr. Lyman Richards, bronchoscopist of the Children's Hospital, Boston, discussed the subject at length. He also spoke in tender detail of his relations with Dr. Chevalier Jackson in his younger days, and also told of several operations that he had performed in this line of work in the Children's Hospital.

After conclusion of Dr. Lyman Richards's remarks, Dr. McCarthy stated that the Plymouth District Medical Society was indebted to Dr. Frederick Murdock, Vice President of the Society (who owns the building in which the meeting was held), for the use of Massasoit Hall free of expense. Too much credit cannot be given Dr. John J. Callahan and President Thomas H. McCarthy for arranging this meeting which will go down in history as one of the best meetings that has ever been arranged in Plymouth County.

THE HARVARD MEDICAL SOCIETY

The Harvard Medical Society held a regular meeting on Tuesday evening, October 29, 1929, at 8:15 o'clock in the Peter Bent Brigham Hospital amphitheatre. After the presentation of cases Dr. S. A. Levine introduced Dr. William G. Lennox, Research Fellow in Neuropathology at the Harvard Medical School, who spoke on "Recent Studies In Epilepsy."

The first case was presented by Dr. Boyd of the surgical service. The patient was a 44 year old business man who had come to the hospital ten days before with a complaint of increasing jaundice for the last six weeks. His past history showed pneumonia ten years before and influenza eight years ago. The present illness began six to seven weeks ago when after dinner he became nauseated and vomited bile-tinged liquid and food. Since then he has had similar attacks. He had clay-colored stools, dark urine, and attacks of acute crampy abdominal pain around and below the umbilicus. In the hospital he was found to be markedly jaundiced, his heart enlarged and with an apical systolic murmur, with a white count of 11,000. Icteric index was 100 and the van den Bergh biphasic. Flat x-rays of the abdomen were negative. Operation revealed a carcinoma of the head of the pancreas and a cholecystoenterostomy was done. His operative recovery was uneventful.

The first case was followed by a series of brief discussions on a certain phase of poliomyelitis and the presentation of a striking example of this subject.

Dr. Aycock first pointed out that the most important event in an attack of poliomyelitis is the paralysis of one or more groups of muscles. The recovery is sometimes striking, especially in the temporary paralysis of the muscles of deglutition where permanent return occurs in the course of a few weeks. Since the main cause of death in poliomyelitis is respiratory paralysis, one is struck by the fact that if these patients could be kept alive by artificial respiration for a time, recovery might ensue. Mr. Philip Drinker has perfected such a machine.

The apparatus was then demonstrated by Dr. Farrell.

Dr. Kramer told the story of the case. The patient was a healthy boy of 21 who spent the summer at a military camp at Lake Champlain. Then he spent three weeks at a camp in Vermont. At no time was he within 20 miles of a case of poliomyelitis. Nine days after reaching home he got up in the morning feeling very ill. The symptoms persisted and a lumbar puncture showed a leucocyte count of eighty. The second day he developed the typical signs of poliomyelitis with a weak left deltoid and quadriceps. The full dose of serum was given. Two days later difficulty in breathing developed and he was brought to the hospital and put in the machine.

Dr. Harrison continued the story in the hospital. The patient was in the machine almost continually for two weeks, being taken out 5-10 minutes twice a day to avoid pneumonia. Out of the tank he complained and was put back in. He was in no pain, took fluids and a soft solid diet well, but had difficulty in sleeping. He continued to improve and at the time of presentation rested comfortably in bed. It is a question whether the tank would help rest him or give him more exercise. Undoubtedly it saved his life.

Dr. Lennox first pointed out that epilepsy is a symptom and not a disease entity. Its precipitating causes may be classed as those inside the brain, either structural or functional, and those outside the brain, among which may be listed the circulation, metabolism, the sympathetic system, endocrine glands, the respiratory and gastro-intestinal tracts. Of many various classes of causes Dr. Lennox undertook to discuss physico-chemical forces.

The first force is that of the water balance in the body. Some have upheld the theory that epilepsy is caused by edema of the brain which in turn is caused by a damming of the Pacchionian bodies. In that case one would expect to find an increase of intracranial pressure, which is not true. Most of the cases studied showed a pressure of between 150-200 mm. Others have said that dehydration helps these patients. In the cases observed in Boston this was not found to be true.

The second physico-chemical factor is the acid-base balance. Acidosis inhibits the attacks. This state may be created by causing a ketosis with special diets, administering acids, re-breathing, and causing an excess formation of lactic acid. On the other hand alkalosis augments the frequency and intensity of the attacks. This condition may result from the administration of alkalis, or by overventilation.

The third force is oxygenation, either of the blood or the tissues. In basal conditions the oxygen saturation was usually within normal limits. In arterial blood some cases showed a degree of anoxemia, but this may have been due to a change in the blood flow and not in the saturation of the tissues. Seizures are easily induced by anoxemia.

The last physico-chemical force considered was the circulation. Attempting to investigate the theory of spasm of cerebral vessels, windows were inserted in the skulls of cats and the effects of various stimuli upon the cerebral vessels studied. It was found that stimulation of the sympathetic narrowed the lumen of the vessels by 8% of their diameters. With a normal oxygen saturation of the blood and a low CO_2 content, the arteries decreased in size; with a high CO_2 content they increased. A similar response was noted with a high oxygen saturation. With a low oxygen saturation and a low CO_2 content, the arteries decreased; with a normal CO_2 content, they showed a slight increase; with a high CO_2 content, a great increase was observed.

The practical points to be gained from all these observations are first, consider the epileptic as a whole and as an individual. His general condition is to be corrected. The acid-base balance may be corrected by more exercise. The posture may be corrected. The peripheral circulation may be helped in any of its abnormalities.

The paper was illustrated by lantern slides and was followed by discussions by Drs. Cobb, Cushing, and Taylor.

HAMPDEN DISTRICT MEDICAL SOCIETY

A meeting of the Hampden District Medical Society was held Oct. 22, 1929, at the Springfield Academy of Medicine.

A splendid program was provided by Louis E. Phaneuf of the Section of Obstetrics and Gynecology, Charles Mongan, Somerville and Frederick Lynch of Boston. The subjects were: "Puerperal Sepsis," "Breech Presentation," and "Version." The two latter papers were illustrated by moving pictures. After adjournment luncheon was served.

NORFOLK DISTRICT MEDICAL SOCIETY

A regular meeting of the Society will be held in the Roxbury Masonic Temple, 171 Warren Street, Roxbury, November 25, 1929 at 8:15 P. M. Tel. Rox. 6089.

PROGRAM

Dr. Roger C. Graves: "Urology from the Standpoint of the General Practitioner."

Discussion: By Dr. W. Richard Ohler.

Collation.

FRANK S. CRUICKSHANK, *Secretary.*

NEW ENGLAND SECTION OF THE AMERICAN ACADEMY OF PHYSIOTHERAPY

The next regular meeting of the New England Section of the American Academy of Physiotherapy will be held at the Square and Compass Club, 448 Beacon Street, Boston, Massachusetts on Wednesday, November 20, 1929 at 8 P. M.

The Speaker will be:

Dr. Charles P. Hutchins, Medical Director of the Rehabilitation Clinic Aetna Life Insurance Company, Syracuse, N. Y.

Subject:

The Place of Physiotherapy in Rehabilitation. Dr. Hutchins was in charge of physiotherapy in the U. S. General Hospital 28, Fort Sheridan,

Illinois, a 4500 bed hospital, during the War and has made physiotherapy his special work since. He will present several moving picture reels. A most profitable and interesting evening is assured.

Dinner at 6 P. M. at the Square and Compass Club. A special menu will be served. It is hoped that many members and guests will make an effort to be present to greet Dr. Hutchins. Dr. Arthur L. Brown will act as Toastmaster. Tickets to the dinner, $1.50. Dress, informal.

If you are coming to the dinner with guests please notify the Secretary before November 18.

BOSTON MEDICAL HISTORY CLUB

The first meeting of the season will be held at the Boston Medical Library on Monday evening, November 18, at 8:15 o'clock.

"The Destruction of the Hunter Manuscripts."—Dr. H. R. Viets.

"The History of Scarlatina"—Dr. A. B. Lyon.

"Why Medical Incunabula?"—James F. Ballard.

JAMES F. BALLARD, *Secretary.*

THE SOUTH END NEIGHBORHOOD MEDICAL CLUB

The next meeting of the South End Neighborhood Medical Club will be held at the Pathological Laboratory, Boston City Hospital, on Tuesday, November 19, 1929, at 12 noon.

The speaker will be Dr. Timothy Leary, Medical Examiner, Suffolk County. His subject will be "Intra-cranial Hemorrhages".

The customary luncheon will be omitted.

NEW ENGLAND ROENTGEN RAY SOCIETY

This meeting will be held Friday, November 15, at the Beth Israel Hospital.

PROGRAM

Some Functional Roentgen Findings of Value in the Treatment of Digestive Disease—10 minutes. Dr. Percy B. Davidson.

Pyelitis of Pregnancy—15 minutes. Dr. E. Granville Crabtree.

The Value of Uterotubography in Gynecological Conditions—20 minutes Dr. Albert A. Shapira.

Lantern Slide Demonstrations of Uterotubograms. Dr. Samuel A. Robins.

Discussion to be opened by Dr. A. K. Paine and Dr. Joseph P. Cohen.

Very truly yours,

T. R. HEALY,

Secretary New England Roentgen Ray Society.

THE AMERICAN PHARMACEUTICAL ASSOCIATION

The semi-annual meeting of the association will be held in the Hotel Washington, Washington, D. C., December 16 and 17, 1929.

The meeting to be held on the first day will be given over largely to problems of executive nature and a general discussion of means and ways of obtaining greater efficiency and economy in distribution.

The second day will be devoted primarily to meeting members of the various government bureaus and departments with which the members of the Association come in contact in the course of their daily activities.

A visit to the Food, Drug and Insecticide Administration, Department of Agriculture and to the Prohibition and Narcotic Divisions of the Treasury Department will occupy the forenoon of December 17.

At the luncheon on this day, Senator George H. Moses, of New Hampshire, President pro tempore of the United States Senate, will be the guest of honor and will address the members. The afternoon will be devoted to addresses by other Government officials and reading and discussion of the reports of the Research Board and the Contact Committee of the Association.

Among the important topics to be discussed in the executive sessions are Publicity, The Proposed Census of dispensing physicians, Institutional Advertising and the "Consult your Family Physician" campaign.

The officers of the Association are:

President: H. Sheridan Baketel, M.D., Jersey City, N. J.

Vice Presidents: Henry Osterman, Seymour, Ind.; E. P. Crowe, Philadelphia, Pa.

Secretary: John G. Searle, Chicago, Ill.

Treasurer: Frank A. Mallett, Des Moines, Iowa.

CLINICAL STAFF OF THE BOSTON DISPENSARY

The November meeting of the Clinical Staff of the Boston Dispensary will be held Tuesday evening, November 19, 1929, at 8:00 P. M. at 25 Bennet Street, under the auspices of the Division of Research.

The program was as follows:

1. "The Diagnosis of Nephrosis." Robert W. Buck, M.D.

2. "The Significance of Axis Deviation in the Human Electrocardiogram." Samuel H. Proger, M.D., David Davis, M.D.

3. "The Occurrence of Normal Blood Pressure Readings in Essential Hypertension." David Ayman, M.D.

4. "The Value of a New Serum Test in Neurosyphilis." Arthur Berk, M.D.

Discussion: Doctors W. Richard Ohler, Paul D. White, Harry C. Solomon, James P. O'Hare and G. Philip Grabfield.

Physicians are cordially invited to attend.

AUGUSTUS RILEY, M.D., *President,*
JOSEPH J. SKIRBALL, M.D., *Secretary.*

WACHUSETT MEDICAL IMPROVEMENT SOCIETY

The regular monthly meeting of the Wachusett Medical Improvement Society was held at the Holden District Hospital. Twenty members were present and one visitor. Supper was served in the usual way at 7:30 P. M. after which the meeting was called to order.

This being the 10th anniversary of the Society Dr.

Washburn was called upon to commemorate the event. Being with Dr. Trask one of the co-founders he was best qualified to tell the members of the humble beginnings of the Society, of its growth, of its reasons for existing and of the advantages it has afforded to its fellows by stimulating their interest in broader knowledge and in better medical work.

Dr. L. D. Riggs of the United States Veterans' Hospital transmitted a message from Dr. H. L. Stickney, inviting the members of the Society to meet at the U. S. Veterans' Hospital on December 4, 1929, and to be his guests at supper. The invitation was cordially extended to the ladies.

The president gave a synopsis of the program for the coming year, and introduced the speaker of the evening, Dr. Edward H. Trowbridge.

Dr. Trowbridge read a paper on the use of the Electro-Surgical Knife in Surgery. The speaker's statements were based on personal observation. He has constantly used the knife for the past two and a half years in all his surgical work. The talk was illustrated by moving picture films of operations performed by the author, who repeatedly called the attention of the physicians to the remarkably small amount of bleeding, thus decreasing considerably the sponging and the trauma which necessarily results from the clamping of bleeding points with the hemostatic forceps.

Discussion followed.

The meeting was adjourned at 10:30 P. M.

PAUL DUFAULT, *Secretary-Treasurer.*

SOCIETY MEETINGS, CONGRESSES AND CONFERENCES

1929-1930—Massachusetts Dietetic Association. Complete schedule appears on page 852, issue of October 24.

November 14—Massachusetts General Hospital. Detailed notice appears on page 964, issue of November 7.

November 15—The New England Roentgen Ray Society. Detailed notice appears on page 1019.

November 18—Boston Medical History Club. Complete notice appears on page 1019.

November 19—The South End Neighborhood Medical Club. Detailed notice appears on page 1019.

November 19—Clinical Staff of the Boston Dispensary. Detailed notice appears on page 1019.

November 20—New England Section of the American Academy of Physiotherapy. Complete notice appears on page 1018.

November 21—New England Heart Association. Complete notice appears on page 906, issue of October 31.

December 2-6—The Radiological Society of North America. Complete notice appears on page 149, issue of July 18.

December 16-17—The American Pharmaceutical Association. Complete notice appears on page 1019.

February 10-14, 1930—American College of Physicians. Complete notice appears on page 853, issue of October 24.

March-October, 1930—International Medical Postgraduate Courses in Berlin. Complete notice appears on page 853, issue of October 24.

May 5-10, 1930—The International Congress on Mental Hygiene. Detailed notice appears on page 853, issue of October 24.

September 4-8, 1930—Fifth International Congress on Physiotherapy. Complete notice appears on page 906, issue of October 31.

DISTRICT MEDICAL SOCIETIES

Essex North District Medical Society

January 8, 1930—Wednesday—Semi-annual meeting at Centre Church Vestries, Main Street, corner of Vestry Street, opposite City Hall, Haverhill, (Telephone 548) at 12:30 sharp.

May 1—Thursday—Censors' meeting at Hotel Bartlett, 95 Main Street, Haverhill, (Telephone 3430) at 2 P. M. sharp. Candidates should present their diplomas to the Secretary one week in advance.

May 7—Wednesday—Annual Meeting.

J. FORREST BURNHAM, Secretary.

Essex South District Medical Society

Wednesday, December 4, 1929—Salem Hospital. Clinic 5 P. M. Dinner 7 P. M. Speaker: Dr. Howard B. Sprague, of Massachusetts General Hospital Heart Clinic. Subject: "Recent Progress in the Diagnosis and Treatment of Heart Disease." General Discussion from the floor.

Wednesday, January 8, 1930—At the Beverly Hospital. Clinic 5 P. M. Dinner 7 P. M. Speaker: Dr. Harry H. Germain, Boston. Subject: "Some Aspects of Cancer of the Colon." General Discussion from the floor.

Wednesday, February 5, 1930—Council Meeting, Boston.

Wednesday, February 12, 1930—Danvers State Hospital, Hathorne. Clinic 5 P. M. Dinner 7 P. M. Speakers: Dr. Winfred Overholser. Subject: "Criminology and Psychiatry." Mr. Clark, District Attorney for Essex County, who will speak on the legal aspect.

Wednesday, March 5, 1930—Lynn Hospital. Clinic 5 P. M. Dinner 7 P. M. Speaker: Dr. R. S. Titus, Boston. Subject: "Some Aspects of Caesarean Section," illustrated with Moving Pictures. General Discussion from the floor.

Wednesday, April 2, 1930—Hotel Hawthorne, Salem. Dinner at 7 P. M. Speaker: Dr. Timothy Leary, Boston. Subject: "Diagnostic and Therapeutic Pitfalls in Intracranial Pyogenic Infections."

Thursday, May 1, 1930—Censors meet for Examination of Candidates at Salem Hospital at 3:30 P. M.

Tuesday, May 13, 1930—Annual Meeting. The Tavern, Gloucester. Speaker: Dr. C. Macfie Campbell, Director of Boston Psychopathic Hospital. Subject to be announced later. Ladies invited. Dancing.

R. E. STONE, M.D., Secretary.

Franklin District Medical Society

The meetings of the Franklin District Medical Society will be held at the Weldon Hotel, Greenfield, Mass., on the second Tuesdays of January, March and May, at 11 A. M.

CHARLES MOLINE, Secretary.

Hampshire District Medical Society

The meetings of Hampshire District will occur at 11 A. M. the second Wednesdays of December, February, and April and will be held at the Dickinson Hospital, Northampton. The annual meeting will be held in May.

LUTHER O. WHITMAN, M.D., Secretary.

Middlesex East District Medical Society

January 15—At Boston, Harvard Club.
March 12—At Melrose.
May 13—At Unicorn Country Club.

ALLAN R. CUNNINGHAM, Secretary.

Middlesex South District Medical Society

April 16, 1930—Annual meeting. Commander Hotel, Cambridge. Program to be announced.

Winter meetings—jointly with Suffolk District.

May, 1930—Censors' meeting.

ALEXANDER A. LEVI, Secretary.

Norfolk District Medical Society

November 25—Roxbury Masonic Temple. Detailed notice appears on page 1018.

January 27, 1930—Roxbury Masonic Temple. "Recent Advances in Diagnosis and Treatment of Syphilis." The speakers will be Drs. C. Morton Smith and Austin W. Cheever.

February 24, 1930—Roxbury Masonic Temple, 8:00. Dr. Edwin H. Place has been invited to read a paper and has been given carte blanche in the selection of his subject.

March 24, 1930—Roxbury Masonic Temple. Dr. Burton E. Hamilton. Subject to be announced.

May 6, 1930—Annual Meeting. Program to be announced.

The Censors meet for the examination of candidates, May 1, 1930, in the Roxbury Masonic Temple, 171 Warren Street, Roxbury, at 4:00 P. M.

Applications must be in the hands of the Secretary at least one week previous to date of examination.

Owing to inability to engage the Roxbury Masonic Apartments on the last Tuesday of the month, such meetings as will be held in the Roxbury Temple will be held Monday evenings as per the dates above.

Suffolk District Medical Society

November 20—See page 1008.

FRANK S. CRUICKSHANK,
Secretary, Norfolk District Medical Society.